

Refine Search

Search Results -

Terms	Documents
mount-jr-david-b.in.	1

Database:

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 US Patents Full-Text Database
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 EPO Abstracts Database
 JPO Abstracts Database
 Derwent World Patents Index
 IBM Technical Disclosure Bulletins

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DATE: Tuesday, February 19, 2008 [Purge Queries](#) [Printable Copy](#) [Create Case](#)

<u>Set Name</u> side by side	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u> result set
<i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=ADJ</i>			
<u>L11</u>	mount-jr-david-b.in.	1	<u>L11</u>
<u>L10</u>	mount-david-b.in.	4	<u>L10</u>
<u>L9</u>	romero-michael-f.in.	6	<u>L9</u>
<u>L8</u>	mount-david-bin.	0	<u>L8</u>
<u>L7</u>	slc26a7	12	<u>L7</u>
<u>L6</u>	sl26a7	0	<u>L6</u>
<u>L5</u>	solute carrier 26\$	4	<u>L5</u>
<u>L4</u>	solute carrier 26A7	0	<u>L4</u>
<u>L3</u>	L2	0	<u>L3</u>
<i>DB=USPT; PLUR=YES; OP=ADJ</i>			
<u>L2</u>	slc26A7	0	<u>L2</u>
<u>L1</u>	6703495.pn.	1	<u>L1</u>

END OF SEARCH HISTORY

\$

FILE 'MEDLINE'
FILE 'JAPIO'
FILE 'BIOSIS'
FILE 'SCISEARCH'

FILE 'WPIDS'
FILE 'CAPLUS'

FILE 'EMBASE'

=> s slc26a7 or solute carrier# 26?
TERM '26?' EXCEEDED TRUNCATION LIMITS - SEARCH ENDED
You have entered a truncated stem which occurs in too many terms.
Make the stem longer and try again. For example, if your original
term was 'degr?' to search for variations and the abbreviation for
'degradation', you could replace it with the expression '(degrdn OR
degrad?)'. If your search term was numeric, e.g., 'C>5', reduce the
size of the range.

=> s slc26a7 or solute carrier# 26a7
L1 134 SLC26A7 OR SOLUTE CARRIER# 26A7

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2 56 DUP REM L1 (78 DUPLICATES REMOVED)

=> d abs ibib 1-56 l1

L1 ANSWER 1 OF 134 MEDLINE on STN
AB Background/Aims: Intercalated cells (ICs) of the kidney collecting
duct
are rich in carbonic anhydrase II (CAII), which facilitates proton
and
bicarbonate transport. Bicarbonate secretion is mediated via
Pendrin
(Slc26a4), which is expressed on the apical membrane of B-ICs and
nonA-nonB ICs in the cortical collecting ducts (CCD). Bicarbonate
absorption is mediated via anion exchanger 1 (AE1-Slc4a1) in the
CCD and
via AE1 and possibly ***Slc26a7*** in the OMCD. Both
exchangers are
expressed on the basolateral membrane of A-ICs. The aim of this
study was
to examine the expression of pendrin, ***Slc26a7***, and AE1 in
the
kidneys of CAII-deficient (CAR2-null) mice. Methods: For the
expression
studies, we used real-time RT-PCR, Northern hybridization,
immunolabeling,
and immunoblotting. Results: Pendrin mRNA expression was reduced
63%
along with decreased pendrin immunolabeling in the cortex of CAR2-
null
mice present predominantly in nonA-nonB ICs. ***Slc26a7***
mRNA
expression was decreases by 73% and ***Slc26a7***
immunolabeling,
present in A-ICs, severely reduced in the outer medulla of CAR2-
null mice.

AE1 mRNA expression was decreased to a similar degree (62%) along with reduced AE1 immunolabeling. The expression of aquaporin 2 (AQP2) water channel, exclusively present in principal cells of the collecting duct, was comparable in the wild type and CAR2-null mice. Conclusion: CAII deficiency results in a significant decrease in the gene and protein expression of bicarbonate transport proteins from Slc26 gene family - Slc26a4 (pendrin) and ***Slc26a7***. These results emphasize the

critical role of CAII for the maintenance of the intercalated cell phenotype. Copyright (c) 2008 S. Karger AG, Basel.

ACCESSION NUMBER: 2008054929 IN-PROCESS <<LOGINID::20080219>>

DOCUMENT NUMBER: PubMed ID: 18209476

TITLE: Decreased Expression of Slc26a4 (Pendrin) and ***Slc26a7*** in the Kidneys of Carbonic

Anhydrase

II-Deficient Mice.

AUTHOR: Sun Xuming; Soleimani Manoocher; Petrovic Snezana

CORPORATE SOURCE: Department of Medicine, University of Cincinnati, Cincinnati, OH., USA.

SOURCE: Cellular physiology and biochemistry : international journal of experimental cellular physiology,

biochemistry,

and pharmacology, (2008) Vol. 21, No. 1-3, pp. 95-

108.

Electronic Publication: 2008-01-16.

Journal code: 9113221. ISSN: 1015-8987.

PUB. COUNTRY: Switzerland

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: NONMEDLINE; IN-DATA-REVIEW; IN-PROCESS; NONINDEXED; Priority Journals

ENTRY DATE: Entered STN: 23 Jan 2008

Last Updated on STN: 23 Jan 2008

L1 ANSWER 2 OF 134 MEDLINE on STN

AB To gain insight into specific gene expression in the gastrointestinal (GI)

tract of preterm infants, we adapted a method to isolate exfoliated epithelial cells. Gastric residual fluid aspirates (n = 89) or

stool

samples (n = 10) were collected from 96 neonates (gestational age,

24-36

wk). Cells were characterized by microscopic observation,

cytokeratin-18

immunodetection, and expression of transcripts. The human origin of

cellular DNA was confirmed by amplification of specific X and Y chromosome

sequences. Isolation yielded 100-500 cells per sample for gastric aspirates (n = 8) and 10-20 cells for fecal samples (n = 5).

Epithelial

origin was confirmed by immunodetection of cytokeratin 18.

Analyses of

reverse transcribed products, using two independent methods, from

15

gastric fluid and two stool samples showed that 18S-rRNA and transcripts of beta-actin, glyceraldehyde-3-phosphate dehydrogenase (gapdh), and period1 were in quantities corresponding to at least 10 cells. On 59 aspirates, we found beta-actin transcripts (all but one), cytokeratin 18 (eight positive of eight samples), SLC26-A7-1 (13 positive of 19 samples), period2 (17 positive of 17 samples), and clock (25 positive of 26 samples). Exfoliated cells can be recovered from gastric aspirates and fecal samples and serve as a tool to investigate the impact of therapeutic

and nutritional regimens on the maturation of GI functions.

ACCESSION NUMBER: 2007716218 MEDLINE <<LOGINID::20080219>>

DOCUMENT NUMBER: PubMed ID: 17805197

TITLE: Recovery of exfoliated cells from the gastrointestinal

tract of premature infants: a new tool to perform "noninvasive biopsies?".

AUTHOR: Kaeffer Bertrand; des Robert Clotilde; Alexandre-Gouabau

Marie-Cecile; Pagniez Anthony; Legrand Arnaud;

Amarger

Valerie; Kuster Alice; Piloquet Hugues; Champ

Martine; le

Huerou-Luron Isabelle; Roze Jean-Christophe

CORPORATE SOURCE: UMR-1280, Physiologie des Adaptations Nutritionnelles,

F-44093 Nantes Cedex 1, France..

Bertand.Kaeffer@nantes.inra.fr

SOURCE: Pediatric research, (2007 Nov) Vol. 62, No. 5, pp. 564-9.

Journal code: 0100714. ISSN: 0031-3998.

PUB. COUNTRY: United States

DOCUMENT TYPE: (EVALUATION STUDIES)

Journal; Article; (JOURNAL ARTICLE)

(RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200802

ENTRY DATE: Entered STN: 11 Dec 2007

Last Updated on STN: 6 Feb 2008

Entered Medline: 5 Feb 2008

L1 ANSWER 3 OF 134 MEDLINE on STN

AB In the present study, the effect of potassium depletion on the expression

of acid-base transporters in the collecting duct was examined.

Toward

this end rats were fed a potassium-free diet for 3 weeks.

Thereafter, the

expression of the basolateral chloride/bicarbonate exchangers AE1

and

SLC26A7 and the apical H(+)-ATPase was examined by northern

hybridization, immunoblot analysis and immunofluorescence labelling. The

mRNA expression of AE1 increased by a robust approximately 500% in

the cortex and approximately 70% in the outer medulla, which translated into a huge increase in AE1 protein abundance in the cortex and a moderate increase in the outer medulla in K-depletion. The mRNA expression of

SLC26A7 did not change significantly but its protein abundance showed a robust increase in the outer medulla. The expression of ***SLC26A7*** remained undetected in the cortex in K-depleted rats. The post translational increase in ***SLC26A7*** membrane abundance in potassium depletion was recapitulated in vitro using epitope-tagged ***SLC26A7***. H(+)-ATPase displayed enhanced apical plasma membrane immunoreactivity in the OMCD in K-depletion. We suggest that the up-regulation of ***SLC26A7*** and AE1 on the basolateral membrane of A-intercalated cells in the OMCD and CCD, respectively, along with H(+)-ATPase on the apical membrane, contributes to enhanced bicarbonate

absorption in the collecting duct in K-depletion.
ACCESSION NUMBER: 2007703928 IN-PROCESS <<LOGINID::20080219>>
DOCUMENT NUMBER: PubMed ID: 17804457
TITLE: Regulation of the basolateral chloride/base exchangers AE1

and ***SLC26A7*** in the kidney collecting duct in potassium depletion.

AUTHOR: Barone Sharon; Amlal Hassane; Kujala Minna; Xu Jie; Karet

Fiona; Blanchard Ann; Kere Juha; Soleimani Manoocher
CORPORATE SOURCE: Department of Medicine, University of Cincinnati, 231

Albert Sabin Way, MSB G259, Cincinnati, OH 45267-0585,

USA.. manoocher.soleimani@uc.edu
SOURCE: Nephrology, dialysis, transplantation : official publication of the European Dialysis and Transplant Association - European Renal Association, (2007 Dec)

Vol. 22, No. 12, pp. 3462-70. Electronic Publication: 2007-09-05.

Journal code: 8706402. ISSN: 0931-0509.

PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: NONMEDLINE; IN-DATA-REVIEW; IN-PROCESS; NONINDEXED; Priority Journals

ENTRY DATE: Entered STN: 29 Nov 2007

Last Updated on STN: 10 Dec 2007

L1 ANSWER 4 OF 134 MEDLINE on STN

AB PURPOSE OF REVIEW: The multifunctional anion exchanger family (Slc26)

encompasses 11 identified genes, but only 10 encode real proteins (Slc26a10 is a pseudogene). Most of the Slc26 proteins function primarily as anion exchangers, exchanging sulfate, iodide, formate, oxalate, hydroxyl ion, and bicarbonate anions, whereas other Slc26 proteins

function as chloride ion channels or anion-gated molecular motors.

The aim of this review is to present recent studies on the molecular function of the Slc26 family and its role in renal physiology and pathophysiology.

RECENT FINDINGS: In proximal tubules, Slc26a1 (Sat-1) mediates sulfate and oxalate transport across the basolateral membrane, while Slc26a6 (CFEX, Pat-1) mediates a variety of anion exchange at the apical membrane to facilitate transcellular sodium chloride absorption. Targeted deletion of murine Slc26a6 leads to intestinal hyperabsorption of oxalate, hyperoxaluria, and kidney stones. Slc26a4 (pendrin) and ***Slc26a7*** are expressed in intercalated cells, and are involved in acid-base homeostasis and blood pressure regulation. Messenger RNA for Slc26a2, Slc26a9, and Slc26a11 is also present in the kidney, yet the roles of these family members in renal physiology or pathophysiology are not clear.

SUMMARY: Members of this multifunctional anion transporter family play evolving roles in the etiology of nephrolithiasis (Slc26a6) and hypertension (Slc26a4 and Slc26a6). Other Slc26 family members (Slc26a2, Slc26a9, Slc26a11) express mRNA in the kidney but their roles in renal physiology are not yet known.

ACCESSION NUMBER: 2007540538 MEDLINE <<LOGINID::20080219>>
DOCUMENT NUMBER: PubMed ID: 17693766
TITLE: Renal physiology of SLC26 anion exchangers.
AUTHOR: Sindic Aleksandra; Chang Min-Hwang; Mount David B; Romero Michael F
CORPORATE SOURCE: Physiology and Biomedical Engineering, Mayo Clinic College of Medicine, Rochester, Minnesota 55905, and Renal Division, Brigham and Women's Hospital, Boston, Massachusetts, USA.
CONTRACT NUMBER: DK056218 (United States NIDDK)
DK57708 (United States NIDDK)
SOURCE: Current opinion in nephrology and hypertension, (2007 Sep) Vol. 16, No. 5, pp. 484-90. Ref: 64
Journal code: 9303753. ISSN: 1062-4821.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200711
ENTRY DATE: Entered STN: 18 Sep 2007
Last Updated on STN: 10 Nov 2007
Entered Medline: 9 Nov 2007

L1 ANSWER 5 OF 134 MEDLINE on STN
 AB AIMS: Anions have an important role in the regulation of airway surface liquid (ASL) volume, viscosity and pH. However, functional localization and regulation of anion exchangers (AEs) have not been clearly described. The aim of this study was to investigate the regulation of AE mRNA expression level in accordance with mucociliary differentiation and the functional expression of AEs cultured normal human nasal epithelial (NHNE) cells. METHODS: Nasal mucosal specimens from three patients are obtained and serially cultured cells are subjected to morphological examinations, RT-PCR, Western blot analysis and immunocytochemistry. AE activity is assessed by pHi measurements. RESULTS: Expression of ciliated cells on the apical membrane and expression of MUC5AC, a marker of mucous differentiation, increased with time. AE2 and SLC26A4 mRNA expression decreased as mucociliary differentiation progressed, and AE4, ***SLC26A7*** and SLC26A8 mRNA expression increased on the 14th and 28th day after confluence. Accordingly, AE4 protein expression also progressively increased. AE activity in 100 mM K(+) buffer solutions was nearly twofold higher than that in 5 mM K(+) buffer solutions. Moreover, only luminal AE activity increased about fourfold over the control in the presence of 5 microM forskolin. In the presence of 100 microM adenosine-5'-triphosphate (ATP) which evokes intracellular calcium signalling through activation of purinergic receptors, only luminal AE activity was again significantly increased. On the other hand, 500 microM 4,4'-diisothiocyanostilbene-2,2'-disulfonic acid (DIDS), an inhibitor of most SLC4 and SLC26AE isoforms, nearly abolished AE activity in both luminal and basolateral membranes. We found that AE activity was affected by intracellular cAMP and calcium signalling in the luminal membrane and was DIDS-sensitive in both membranes of cultured NHNE cells.

CONCLUSION:
 Our findings through molecular and functional studies using cultured NHNE cells suggest that AEs may have an important role in the regulation of ASL.

ACCESSION NUMBER: 2007533083 IN-PROCESS <<LOGINID::20080219>>
 DOCUMENT NUMBER: PubMed ID: 17635413
 TITLE: Molecular and functional expression of anion exchangers in cultured normal human nasal epithelial cells.
 AUTHOR: Shin J-H; Son E J; Lee H S; Kim S J; Kim K; Choi J

Y; Lee M

G; Yoon J-H

CORPORATE SOURCE: The Airway Mucus Institute, Yonsei University
College of

Medicine, Seoul, Korea.

SOURCE: Acta physiologica (Oxford, England), (2007 Oct) Vol.
191,

No. 2, pp. 99-110. Electronic Publication: 2007-07-

17.

Journal code: 101262545. ISSN: 1748-1708.

PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English

FILE SEGMENT: NONMEDLINE; IN-PROCESS; NONINDEXED; Priority
Journals

ENTRY DATE: Entered STN: 13 Sep 2007

Last Updated on STN: 8 Dec 2007

L1 ANSWER 6 OF 134 MEDLINE on STN

AB To date three potential candidates for parietal cell basolateral Cl
(-)

entry have been described: the highly 4,4'-
diisothiocyanatostilbene-2,2'-
disulfonic acid (DIDS)-sensitive [Formula: see text] exchanger AE2,
the

[Formula: see text] and lowly DIDS-sensitive ***SLC26A7***
protein,

and the Na(+)-2Cl(-)K(+) cotransporter (NKCC1). In this study we
investigate the contribution of these pathways to secretagogue
stimulated

acid secretion. Individually hand-dissected rat gastric glands
were

microfluorimetrically monitored for Cl(-) influx and pH(i) changes.
Transporter activity was determined by varying ion content and
through the

use of pharmacological inhibitors. Expression of ***SLC26A7***
in rat

parietal cells was shown by immunohistochemistry and Western blot.
SLC26A7 was inhibited by 5-Nitro-2-(3-phenylpropyl-amino)
benzoic

acid (NPPB) (100 microM) in the Xenopus laevis oocyte expression
system.

Cl(-) influx in parietal cells was enhanced by histamine, depended
partially on endogenous [Formula: see text] synthesis and
completely on

extracellular Na(+). Removal and subsequent readdition of Cl(-)
revealed

a low and a high DIDS-sensitive [Formula: see text] extrusion
system

contributing to Cl(-) uptake. At acidic pH(i), however, H(+)
extrusion

via the H(+),K(+)-ATPase depending on Cl(-) uptake was abolished
only in.

the presence of 100 microM (NPPB) and at high (250 microM) DIDS
concentration. There was no effect of the NKCC inhibitor
bumetanide on

stimulated H(+) extrusion. These results would be compatible with
SLC26A7 as a Cl(-) uptake system under histamine
stimulation.

ACCESSION NUMBER: 2007505078 MEDLINE <<LOGINID::20080219>>

DOCUMENT NUMBER: PubMed ID: 17404755
 TITLE: ***SLC26A7*** can function as a chloride-loading mechanism in parietal cells.
 AUTHOR: Kosiek Ortrud; Busque Stephanie M; Foller Michael; Shcheynikov Nikolay; Kirchhoff Philipp; Bleich Markus;
 CORPORATE SOURCE: Muallem Shmuel; Geibel John P
 Department of Surgery, Yale University School of Medicine,
 BML 265, 310 Cedar Street, New Haven, CT, 06520, USA.
 CONTRACT NUMBER: DE12309 (United States NIDCR)
 DK50230 (United States NIDDK)
 SOURCE: Pflugers Archiv : European journal of physiology, (2007
 Sep) Vol. 454, No. 6, pp. 989-98. Electronic
 Publication: 2007-04-03.
 Journal code: 0154720. ISSN: 0031-6768.
 PUB. COUNTRY: Germany: Germany, Federal Republic of
 DOCUMENT TYPE: (IN VITRO)
 Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200801
 ENTRY DATE: Entered STN: 30 Aug 2007
 Last Updated on STN: 29 Jan 2008
 Entered Medline: 24 Jan 2008

L1 ANSWER 7 OF 134 MEDLINE on STN
 AB Uptake of SO(4) (2-) by articular chondrocytes is an essential step in the pathway for sulphation of glycosaminoglycans (GAGs), with mutations in SO(4) (2-) transport proteins resulting in abnormalities of skeletal growth. In the present study, the transporters mediating SO(4) (2-) transport in bovine articular chondrocytes have been characterized. Expression of candidate transporters was determined using RT-PCR, while SO(4) (2-) transport was measured in radioisotope flux experiments. RT-PCR experiments showed that bovine articular chondrocytes express three transporters known to transport SO(4) (2-): AE2 (SLC4a2), DTDST (SLC26a2), and SLC26a11. Other transporters--NaS-1 (SLC13a1), SAT-1 (SLC26a1), DRA (SLC26a3), SLC26a6 (PAT1), ***SLC26a7***, SLC26a8 (Tat-1), and SLC26a9--were, however, not detected. In functional experiments, SO(4) (2-) uptake was temperature-sensitive, inhibited by 60% by DIDS (50 microM) and exhibited saturation kinetics, with a K(m) value of 16 mM. Uptake was also inhibited at alkaline extracellular pH. In further experiments, a K(i) value for DIDS inhibition of SO(4) (2-) efflux of 5 microM was recorded. A DIDS-sensitive component of SO(4) (2-) efflux

persisted in solutions lacking Cl(-) ions. These data are interpreted as evidence for the preferential operation of carrier-mediated exchange of

SO(4) (2-) for Cl(-), while an alternative SO(4) (2-)-OH(-) exchange mode

is also possible.

ACCESSION NUMBER: 2007491251 MEDLINE <<LOGINID::20080219>>

DOCUMENT NUMBER: PubMed ID: 17474136

TITLE: Characterization of sulphate transporters in isolated

bovine articular chondrocytes.

AUTHOR: Meredith David; Gehl Katharina A; Seymour John; Ellory J

Clive; Wilkins Robert J

CORPORATE SOURCE: Department of Physiology, Anatomy and Genetics, Sherrington

Building, Parks Road, Oxford, OX1 3PT, UK.

SOURCE: Journal of orthopaedic research : official publication of

the Orthopaedic Research Society, (2007 Sep) Vol.

25, No.

9, pp. 1145-53.

Journal code: 8404726. ISSN: 0736-0266.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200709

ENTRY DATE: Entered STN: 23 Aug 2007

Last Updated on STN: 28 Sep 2007

Entered Medline: 27 Sep 2007

L1 ANSWER 8 OF 134 MEDLINE on STN

AB Association of some plasma membrane bicarbonate transporters with carbonic

anhydrase enzymes forms a bicarbonate transport metabolon to facilitate

metabolic CO(2)-HCO(3)(-) conversions and coupled HCO(3)(-) transport.

The transmembrane carbonic anhydrase, CAIX, with its extracellular catalytic site, is highly expressed in parietal and other cells of gastric

mucosa, suggesting a role in acid secretion. We examined in transfected

HEK293 cells the functional and physical interactions between CAIX and the

parietal cell Cl(-)/HCO(3)(-) exchanger AE2 or the putative Cl(-)/HCO(3)(-) exchanger ***SLC26A7***. Coexpression of CAIX increased AE2 transport activity by 28 +/- 7% and also activated transport

mediated by AE1 and AE3 (32 +/- 10 and 37 +/- 9%, respectively).

In

contrast, despite a transport rate comparable to that of AE3, coexpressed

CAIX did not alter transport associated with ***SLC26A7***.

The

CAIX-associated increase of AE2 activity did not result from altered AE2

expression or cell surface processing. CAIX was

coimmunoprecipitated with
the coexpressed SLC4 polypeptides AE1, AE2, and AE3, but not with
SLC26A7 . GST pull-down assays with a series of domain-
deleted
forms of CAIX revealed that the catalytic domain of CAIX mediated
interaction with AE2. AE2 and CAIX colocalized in human gastric
mucosa,
as indicated by coimmunofluorescence. This is the first example of
a
functional and physical interaction between a bicarbonate
transporter and
a transmembrane carbonic anhydrase. We conclude that CAIX can bind
to
some Cl(-)/HCO(3)(-) exchangers to form a bicarbonate transport
metabolon.

ACCESSION NUMBER: 2007457380 MEDLINE <<LOGINID::20080219>>
DOCUMENT NUMBER: PubMed ID: 17652430
TITLE: Interactions of transmembrane carbonic anhydrase,
CAIX,
with bicarbonate transporters.
AUTHOR: Morgan Patricio E; Pastorekova Silvia; Stuart-Tilley
Alan
CORPORATE SOURCE: K; Alper Seth L; Casey Joseph R
Membrane Protein Research Group, Dept of Physiology,
University of Alberta, Edmonton, Alberta, Canada.
CONTRACT NUMBER: CA101942 (United States NCI)
DK34854 (United States NIDDK)
DK43495 (United States NIDDK)
SOURCE: American journal of physiology. Cell physiology,
(2007 Aug)
Publication: Vol. 293, No. 2, pp. C738-48. Electronic
2007-07-25.
Journal code: 100901225. ISSN: 0363-6143.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200709
ENTRY DATE: Entered STN: 7 Aug 2007
Last Updated on STN: 21 Sep 2007
Entered Medline: 20 Sep 2007

L1 ANSWER 9 OF 134 MEDLINE on STN
AB Appropriate intraluminal microenvironment in the epididymis is
essential
for maturation of sperm. To clarify whether the anion transporters
SLC26A2, SLC26A6, ***SLC26A7*** , and SLC26A8 might participate
in
generating this proper intraluminal milieu, we studied the
localization of
these proteins in the human efferent and the epididymal ducts by
immunohistochemistry. In addition, immunohistochemistry of several
SLC26-interacting proteins was performed: the Na(+)/H(+) exchanger
3
(NHE3), the Cl(-) channel cystic fibrosis transmembrane conductance
regulator (CFTR), the proton pump V-ATPase, their regulator Na(+)/H
(+)
exchanger regulating factor 1 (NHERF-1), and carbonic anhydrase II

(CAII).

Our results show that SLC26A6, CFTR, NHE3, and NHERF-1 are co-expressed on the apical side of the nonciliated cells, and SLC26A2 appears in the cilia of the ciliated cells in the human efferent ducts. In the epididymal ducts, SLC26A6, CFTR, NHERF-1, CAII, and V-ATPase (B and E subunits) were co-localized to the apical mitochondria rich cells, while ***SLC26A7*** was expressed in a subgroup of basal cells. SLC26A8 was not found in the structures studied. This is the first study describing the localization of SLC26A2, A6 and A7, and NHERF-1 in the efferent and the epididymal ducts. Immunolocalization of human CFTR, NHE3, CAII, and V-ATPase in these structures differs partly from previous reports from rodents. Our findings suggest roles for these proteins in male fertility, either independently or through interaction and reciprocal regulation with co-localized proteins shown to affect fertility, when disrupted.

ACCESSION NUMBER: 2007344787 MEDLINE <<LOGINID::20080219>>
DOCUMENT NUMBER: PubMed ID: 17504921
TITLE: Expression of ion transport-associated proteins in human efferent and epididymal ducts.
AUTHOR: Kujala Minna; Hihnala Satu; Tienari Jukka; Kaunisto Kari;
Hastbacka Johanna; Holmberg Christer; Kere Juha; Hoglund Pia
CORPORATE SOURCE: Department of Medical Genetics, University of Helsinki, Helsinki, Finland.. minna.kujala@helsinki.fi
SOURCE: Reproduction (Cambridge, England), (2007 Apr) Vol. 133, No. 4, pp. 775-84.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200709
ENTRY DATE: Entered STN: 12 Jun 2007
Last Updated on STN: 27 Sep 2007
Entered Medline: 26 Sep 2007

L1 ANSWER 10 OF 134 MEDLINE on STN
AB Human red cell anion exchanger AE1 (band 3) is an electroneutral Cl-HCO3-exchanger with 12-14 transmembrane spans (TMs). Previous work using *Xenopus* oocytes has shown that two co-expressed fragments of AE1 lacking TMs 6 and 7 are capable of forming a stilbene disulphonate-sensitive (36)Cl-influx pathway, reminiscent of intact AE1. In the present

study,
 we create a single construct, AE1Delta(6: 7), representing the
 intact
 protein lacking TMs 6 and 7. We expressed this construct in
 Xenopus
 oocytes and evaluated it employing a combination of two-electrode
 voltage
 clamp and pH-sensitive microelectrodes. We found that, whereas
 AE1Delta(6: 7) has some electroneutral Cl-base exchange activity,
 the
 protein also forms a novel anion-conductive pathway that is blocked
 by
 DIDS. The mutation Lys(539)Ala at the covalent DIDS-reaction site
 of AE1
 reduced the DIDS sensitivity, demonstrating that (1) the conductive
 pathway is intrinsic to AE1Delta(6: 7) and (2) the conductive
 pathway has
 some commonality with the electroneutral anion-exchange pathway.
 The
 conductance has an anion-permeability sequence: NO3- approximately
 I- >
 NO2- > Br- > Cl- > SO4(2-) approximately HCO3- approximately
 gluconate-
 approximately aspartate- approximately cyclamate-. It may also
 have a
 limited permeability to Na+ and the zwitterion taurine. Although
 this
 conductive pathway is not a usual feature of intact mammalian AE1,
 it
 shares many properties with the anion-conductive pathways intrinsic
 to two
 other Cl-HCO3- exchangers, trout AE1 and mammalian ***SLC26A7***

ACCESSION NUMBER: 2007288860 MEDLINE <<LOGINID::20080219>>
 DOCUMENT NUMBER: PubMed ID: 17317744
 TITLE: A conductive pathway generated from fragments of the
 human
 red cell anion exchanger AE1.
 AUTHOR: Parker Mark D; Young Mark T; Daly Christopher M;
 Meech
 Robert W; Boron Walter F; Tanner Michael J A
 CORPORATE SOURCE: Department of Biochemistry, University of Bristol,
 University Walk, Bristol, BS8 1TD, UK..
 mark.parker@yale.edu
 CONTRACT NUMBER: NS18400 (United States NINDS)
 P30-34989
 SOURCE: The Journal of physiology, (2007 May 15) Vol. 581,
 No. Pt
 1, pp. 33-50. Electronic Publication: 2007-02-22.
 Journal code: 0266262. ISSN: 0022-3751.
 PUB. COUNTRY: England: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200707
 ENTRY DATE: Entered STN: 16 May 2007
 Last Updated on STN: 24 Jul 2007
 Entered Medline: 23 Jul 2007

L1 ANSWER 11 OF 134 MEDLINE on STN
 AB Sulfate is essential for normal cellular function. The kidney plays a major role in sulfate homeostasis. Sulfate is freely filtered and then undergoes net reabsorption in the proximal tubule. The apical membrane Na(+)/sulfate cotransporter NaS1 (SLC13A1) has a major role in mediating proximal tubule sulfate reabsorption, as demonstrated by the findings of hyposulfatemia and hypersulfaturia in Nas1-null mice. The anion exchanger SAT1 (SLC26A1), the founding member of the SLC26 sulfate transporter family, mediates sulfate exit across the basolateral membrane to complete the process of transtubular sulfate reabsorption. Another member of this family, CFEX (SLC26A6), is present at the apical membrane of proximal tubular cells. It also can transport sulfate by anion exchange, which probably mediates backflux of sulfate into the lumen. Knockout mouse studies have demonstrated a major role of CFEX as an apical membrane Cl(-)/oxalate exchanger that contributes to NaCl reabsorption in the proximal tubule. Several additional SLC26 family members mediate sulfate transport and show some level of renal expression (e.g., SLC26A2, ***SLC26A7***, SLC26A11). Their roles in mediating renal tubular sulfate transport are presently unknown. This paper reviews current data available on the function and regulation of three sulfate transporters (NaS1, SAT1, and CFEX) and their physiological roles in the kidney.

ACCESSION NUMBER: 2007207344 MEDLINE <<LOGINID::20080219>>
 DOCUMENT NUMBER: PubMed ID: 17002596
 TITLE: Specificity and regulation of renal sulfate transporters.
 AUTHOR: Markovich Daniel; Aronson Peter S
 CORPORATE SOURCE: Department of Physiology and Pharmacology, School of Biomedical Sciences, University of Queensland, Brisbane,
 QLD 4072 Australia.. d.markovich@uq.edu.au
 CONTRACT NUMBER: P01-DK17433 (United States NIDDK)
 R01-DK33793 (United States NIDDK)
 SOURCE: Annual review of physiology, (2007) Vol. 69, pp. 361-75.
 Ref: 97
 Journal code: 0370600. ISSN: 0066-4278.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 General Review; (REVIEW)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals

ENTRY MONTH: 200704
ENTRY DATE: Entered STN: 7 Apr 2007
Last Updated on STN: 27 Apr 2007
Entered Medline: 26 Apr 2007

L1 ANSWER 12 OF 134 MEDLINE on STN
AB The majority of the Na⁺ and Cl⁻ filtered by the kidney is reabsorbed in the proximal tubule. In this nephron segment, a significant fraction of Cl⁻ is transported via apical membrane Cl⁻/base exchange: Cl⁻/formate exchange in parallel with Na⁺/H⁺ exchange and H⁺/formate cotransport, and Cl⁻/oxalate exchange in parallel with oxalate/sulfate exchange and Na⁺/sulfate cotransport. Apical membrane Cl⁻-OH⁻ or Cl⁻/HCO₃⁻ exchange has also been observed. NHE3 mediates most if not all apical membrane Na⁺/H⁺ exchange in the proximal tubule. We evaluated SLC26 family members as candidates to mediate proximal tubule Cl⁻/base exchange. We could not detect pendrin (SLC26A4) expression in the proximal tubule, and found no change in transtubular NaCl absorption in pendrin null mice. We did find expression of SLC26A6 (CFEX, PAT1) on the apical membrane of proximal tubule cells, and demonstrated that SLC26A6 is capable of mediating the Cl⁻/base exchange activities described to take place across the brush border membrane. Microperfusion studies on SLC26A6 null mice demonstrated that SLC26A6 is essential for oxalate-dependent NaCl absorption but does not contribute to baseline transport, suggesting it primarily mediates Cl⁻/oxalate exchange rather than Cl⁻-OH⁻ or Cl⁻/HCO₃⁻ exchange in the proximal tubule. Expression of ***SLC26A7*** was also detected on the brush border membrane of proximal tubule cells. Finally, we demonstrated an essential role for the scaffolding protein PDZK1 in apical membrane expression of SLC26A6.

ACCESSION NUMBER: 2006680887 MEDLINE <<LOGINID::20080219>>
DOCUMENT NUMBER: PubMed ID: 17120766
TITLE: Role of SLC26-mediated Cl⁻/base exchange in proximal tubule NaCl transport.
AUTHOR: Aronson Peter S
CORPORATE SOURCE: Department of Medicine, Yale University School of Medicine,
New Haven, CT 06520-8029, USA.
CONTRACT NUMBER: P01-DK17433 (United States NIDDK)
R01-DK37933 (United States NIDDK)
SOURCE: Novartis Foundation symposium, (2006) Vol. 273, pp. 148-58;

discussion 158-63, 261-4.
 Journal code: 9807767. ISSN: 1528-2511.
 PUB. COUNTRY: England: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200702
 ENTRY DATE: Entered STN: 24 Nov 2006
 Last Updated on STN: 13 Feb 2007
 Entered Medline: 12 Feb 2007

L1 ANSWER 13 OF 134 MEDLINE on STN
 AB SLC26 anion exchangers transport monovalent and divalent anions,
 with a
 diversity of anion specificity and stoichiometry. Our
 microelectrode
 studies indicate that several SLC26 members are electrogenic. We
 reported
 that Slc26a6 functions as a Cl⁻/formate, Cl⁻/oxalate, Cl⁻/OH⁻ and
 electrogenic Cl⁻/nHCO₃⁻ exchanger. Recently, we have also
 confirmed that
 Slc26a7 does not behave as a Cl⁻/HCO₃⁻ exchanger but does
 function
 as an electrogenic anion conductance, perhaps a channel. We have
 also
 cloned murine Slc26a9, which is strongly expressed in the
 respiratory
 tract and stomach. Radioisotope uptakes in Xenopus oocytes
 indicate that
 Slc26a9 is a highly selective anion exchanger, transporting Cl⁻ but
 neither formate, oxalate, nor SO₄²⁻. We also utilized
 electrophysiology
 to voltage clamp (VC) and/or measure intracellular pH (pHi), Cl⁻
 ([Cl⁻]_i),
 and Na⁺ ([Na⁺]_i), in response to various ion replacements. Cl⁻
 removal in
 HCO₃⁻ depolarizes oocytes (to > +60mV), alkalinizes oocytes, and
 decreases
 aCl⁻_i. Slc26a9 thus functions as an electrogenic nCl⁻/HCO₃⁻
 exchanger,
 suggesting a role in pulmonary and gastric HCO₃⁻ secretion and/or
 CO₂
 transport. VC experiments revealed channel-like currents (>10
 microA at
 -60mV and >80 microA at +60mV) mediated by Slc26a9 in the presence
 and
 absence of HCO₃⁻. Our experiments and those of others continue to
 reveal
 additional characteristics and unique roles for this new class of
 electrogenic anion transporters.

ACCESSION NUMBER: 2006680886 MEDLINE <<LOGINID::20080219>>
 DOCUMENT NUMBER: PubMed ID: 17120765
 TITLE: Physiology of electrogenic SLC26 paralogues.
 AUTHOR: Romero Michael F; Chang Min-Hwang; Plata Consuelo;
 Zandi-Nejad Kambiz; Mercado Adriana; Broumand
 Vadjista;
 Sussman Caroline R; Mount David B
 CORPORATE SOURCE: Physiology & Biophysics, Case Western Reserve
 University,
 School of Medicine, Cleveland, OH 44106-4970, USA.

CONTRACT NUMBER: DK-56218 (United States NIDDK)
 DK-60845 (United States NIDDK)
 DK038226 (United States NIDDK)
 DK57708 (United States NIDDK)
 SOURCE: Novartis Foundation symposium, (2006) Vol. 273, pp.
 126-38;
 discussion 138-47, 261-4.
 Journal code: 9807767. ISSN: 1528-2511.
 PUB. COUNTRY: England: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 (RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200702
 ENTRY DATE: Entered STN: 24 Nov 2006
 Last Updated on STN: 13 Feb 2007
 Entered Medline: 12 Feb 2007

L1 ANSWER 14 OF 134 MEDLINE on STN
 AB SLC26 isoforms are members of a large, conserved family of anion
 exchangers that display highly restricted and distinct tissue
 distribution. Cloning experiments have identified the existence of
 10 SLC26 genes or isoforms (SLC26A1-11). The products of all,
 excepting
 SLC26A5 (prestin), function as anion exchangers with versatility
 with
 respect to transported anions. Modes of transport mediated by
 SLC26
 members include the exchange of chloride for bicarbonate, hydroxyl,
 sulfate, formate, iodide, or oxalate with variable specificity.
 Several
 members of SLC26 family mediate chloride-bicarbonate exchange and
 display
 expression in a limited number of tissues including the
 gastrointestinal
 tract and/or kidney, with distinct subcellular (apical or
 basolateral)
 localization. These include SLC26A3 (DRA), SLC26A4 (pendrin),
 SLC26A6
 (PAT1 or CFEX), ***SLC26A7*** and SLC26A9. SLC26A3 and A9 are
 not
 expressed in the kidney but SLC26A4, A6 and A7 are. Genetically
 engineered null mice have highlighted the important role of two
 members of
 the SLC26 family, SLC26A4 and SLC26A6, in homeostatic function in
 kidney
 and/or intestine. In conjunction with expression studies, the
 evolving
 picture points to important roles for SLC26 family in chloride,
 bicarbonate, oxalate or sulfate transport and homeostasis in
 gastrointestinal tract, kidney and several other tissues. This
 review in
 particular focuses on the role and regulation of SLC26A6, A7 and A9
 in the
 kidney and/or gastrointestinal tract.
 ACCESSION NUMBER: 2006680884 MEDLINE <<LOGINID::20080219>>
 DOCUMENT NUMBER: PubMed ID: 17120763
 TITLE: Expression, regulation and the role of SLC26

Cl-/HCO3-

exchangers in kidney and gastrointestinal tract.
AUTHOR: Soleimani Manoocher
CORPORATE SOURCE: Department of Medicine, University of Cincinnati and
Veterans Affairs Medical Center, Cincinnati, OH, USA.
CONTRACT NUMBER: DK 62809 (United States NIDDK)
SOURCE: Novartis Foundation symposium, (2006) Vol. 273, pp.
91-102;
discussion 103-6, 261-4. Ref: 60
Journal code: 9807767. ISSN: 1528-2511.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200702
ENTRY DATE: Entered STN: 24 Nov 2006
Last Updated on STN: 13 Feb 2007
Entered Medline: 12 Feb 2007

L1 ANSWER 15 OF 134 MEDLINE on STN
AB In the late 1990s the SLC26 family of anion exchangers emerged as
the
second, structurally distinct gene family capable of similar
transport
functions as the classical SLC4 or anion exchanger (AE) gene
family. The
observations leading to the characterization of the SLC26 family
were
firmly based on research on rare human diseases and aided by
comparison to
Caenorhabditis elegans. SLC26A1, or rat sulphate/anion transporter
1
(Sat1), was the first gene cloned in mammals, but not characterized
in
humans until the year 2000. Three rare recessive diseases in
humans,
namely diastrophic dysplasia (cartilage disorder resulting in
growth
retardation), congenital chloride diarrhoea (anion exchange
disorder of
the intestine) and Pendred syndrome (deafness with thyroid
disorder)
turned out to be caused by the highly related genes SLC26A2 (first
called
DTDST), SLC26A3 (first called CLD or DRA) and SLC26A4 (first called
PDS),
respectively. Subsequently, others and our laboratory cloned
prestin, a
cochlear motor protein gene (SLC26A5), a putative pancreatic anion
transporter (SLC26A6), and ***SLC26A7*** -SLC26A11. Some SLC26
family
members show highly specific tissue expression patterns, others are
widely
expressed. The SLC26 exchangers are capable of transporting, with
different affinities, at least the chloride, iodide, sulfate,
bicarbonate,
hydroxyl, oxalate and formate anions, and have distinct anion
specificity

profiles.

ACCESSION NUMBER: 2006680879 MEDLINE <<LOGINID::20080219>>
DOCUMENT NUMBER: PubMed ID: 17120758
TITLE: Overview of the SLC26 family and associated diseases.
AUTHOR: Kere Juha
CORPORATE SOURCE: Department of Biosciences at Novum, Karolinska Institutet,
14157 Huddinge, Sweden.
SOURCE: Novartis Foundation symposium, (2006) Vol. 273, pp. 2-11;
discussion 11-8, 261-4. Ref: 32
Journal code: 9807767. ISSN: 1528-2511.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200702
ENTRY DATE: Entered STN: 24 Nov 2006
Last Updated on STN: 13 Feb 2007
Entered Medline: 12 Feb 2007

L1 ANSWER 16 OF 134 MEDLINE on STN
AB Solute-linked carrier 26 (SLC26) isoforms are members of a large, conserved family of anion exchangers, many of which display highly restricted and distinct tissue distribution. Cloning experiments have identified 10 SLC26 genes or isoforms (SLC26A1-11). Except for SLC26A5 (prestin), all function as anion exchangers with versatility with respect to transported anions. Modes of transport mediated by SLC26 members include the exchange of chloride for bicarbonate, hydroxyl, sulfate, formate, iodide, or oxalate with variable specificity. Other anion exchange modes not involving chloride also have been reported for some of the members of this family. Several members of SLC26 isoforms are expressed in the kidney. These include SLC26A1 (SAT1), SLC26A4 (pendrin), SLC26A6 (putative anion transporter [PAT1] or chloride/formate exchange [CFEX]), ***SLC26A7***, and SLC26A11. Each isoform displays a specific nephron segment distribution with a distinct subcellular localization. Coupled to expression studies and examination of genetically engineered mice deficient in various SLC26 isoforms, the evolving picture points to important roles for the SLC26 family in chloride absorption, vascular volume homeostasis, acid-base regulation, and oxalate excretion in the kidney. This review summarizes recent advances in the identification and characterization of SLC26 family members, with specific emphasis on their distribution and role in kidney physiology. Specifically, the roles of A4 (pendrin), A6 (PAT1), and A7 (PAT2) in chloride homeostasis, oxalate excretion, and acid-base balance

are discussed.

ACCESSION NUMBER: 2006636258 MEDLINE <<LOGINID::20080219>>
DOCUMENT NUMBER: PubMed ID: 17071331
TITLE: SLC26 chloride/base exchangers in the kidney in
health and disease.
AUTHOR: Soleimani Manoocher; Xu Jie
CORPORATE SOURCE: Division of Nephrology and Hypertension, Department
of Medicine, University of Cincinnati, 231 Albert Sabin
Way, Cincinnati, OH 45267, USA..
manoocher.soleimani@uc.edu
CONTRACT NUMBER: DK 62809 (United States NIDDK)
R01 DK 54430 (United States NIDDK)
SOURCE: Seminars in nephrology, (2006 Sep) Vol. 26, No. 5,
pp. 375-85. Ref: 64
Journal code: 8110298. ISSN: 0270-9295.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200702
ENTRY DATE: Entered STN: 31 Oct 2006
Last Updated on STN: 9 Feb 2007
Entered Medline: 8 Feb 2007

L1 ANSWER 17 OF 134 MEDLINE on STN
AB ***SLC26A7*** is a newly identified basolateral Cl(-)/HCO(3)(-) exchanger specific to alpha-intercalated cells of the outer medullary collecting duct (OMCD). The purpose of the present experiments was to examine the expression of ***SLC26A7*** in kidneys of vasopressin-deficient Brattleboro rats before and after treatment with desamino-Cys(1),d-Arg(8)-vasopressin (dDAVP). Brattleboro rats were treated with dDAVP, a vasopressin analog, for 8 days, and their kidneys were examined for the expression of ***SLC26A7***. The expression of ***SLC26A7*** protein, as examined by immunofluorescence, was undetectable in kidneys of Brattleboro rats. However, treatment with dDAVP induced expression of ***SLC26A7*** protein, restoring it to levels observed in normal rats. These results were verified by Western blot analysis. The mRNA expression of ***SLC26A7*** remained unchanged in response to dDAVP. Immunofluorescent labeling demonstrated abundant levels of anion exchanger type 1 in the OMCD of Brattleboro rats and a mild reduction in response to dDAVP. The abundance of H(+)-ATPase was not affected by dDAVP. The increased ***SLC26A7***

expression
directly correlated with enhanced aquaporin-2 expression, which is
proportional to increased interstitial osmolarity in the medulla.
In
conclusion, vasopressin increases the expression of ***SLC26A7***
protein through posttranscriptional mechanisms in the OMCD. The
induction
of ***SLC26A7*** by vasopressin in OMCD cells of Brattleboro
rats is
likely an attempt by cells to regulate their cell volume and
maintain
HCO(3)(-) absorption in a state associated with increased
interstitial
medullary tonicity.

ACCESSION NUMBER: 2006194140 MEDLINE <<LOGINID::20080219>>
DOCUMENT NUMBER: PubMed ID: 16352747
TITLE: Vasopressin induces expression of the Cl-/HCO3-
exchanger
SLC26A7 in kidney medullary collecting
ducts of
Brattleboro rats.
AUTHOR: Petrovic Snezana; Amlal Hassane; Sun Xuming; Karet
Fiona;
Barone Sharon; Soleimani Manoocher
CORPORATE SOURCE: Div. of Nephrology and Hypertension, Dept. of
Medicine,
Univ. of Cincinnati, 231 Albert Sabin Way, MSB 259G,
Cincinnati, OH 45267-0585, USA..
Snezana.Petrovic@uc.edu
CONTRACT NUMBER: DK-62809 (United States NIDDK)
SOURCE: American journal of physiology. Renal physiology,
(2006
May) Vol. 290, No. 5, pp. F1194-201. Electronic
Publication: 2005-12-13.
Journal code: 100901990. ISSN: 0363-6127.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200605
ENTRY DATE: Entered STN: 8 Apr 2006
Last Updated on STN: 23 May 2006
Entered Medline: 22 May 2006

L1 ANSWER 18 OF 134 MEDLINE on STN
AB ***SLC26A7*** is a Cl(-)/HCO(3)(-) exchanger that is expressed
on the
basolateral membrane and in the cytoplasm of two distinct acid-
secreting
epithelial cells: The A-intercalated cells in the kidney outer
medullary
collecting duct and the gastric parietal cells. The intracellular
localization of ***SLC26A7*** suggests the possibility of
trafficking
between cell membrane and intracellular compartments. For testing
this
hypothesis, full-length human ***SLC26A7*** cDNA was fused with
green
fluorescence protein and transiently expressed in MDCK epithelial

cells.
 In monolayer cells in isotonic medium, ***SLC26A7*** showed punctate distribution throughout the cytoplasm. However, in medium that was made hypertonic for 16 h, ***SLC26A7*** was detected predominantly in the plasma membrane. The presence of mitogen-activated protein kinase inhibitors blocked the trafficking of ***SLC26A7*** to the plasma membrane. Double-labeling studies demonstrated the localization of ***SLC26A7*** to the transferrin receptor-positive endosomes.

A chimera that was composed of the amino terminal fragment of ***SLC26A7*** and the carboxyl terminal fragment of SLC26A1, and a C-terminal-truncated ***SLC26A7*** were retained in the cytoplasm in hypertonicity.

In separate studies, ***SLC26A7*** showed predominant localization in plasma membrane in potassium-depleted isotonic medium (0.5 or 2 mEq/L KCl) versus cytoplasmic distribution in normal potassium isotonic medium (4 mEq/L). It is concluded that ***SLC26A7*** is present in endosomes, and its targeting to the basolateral membrane is increased in hypertonicity and potassium depletion. The trafficking to the cell surface suggests novel functional upregulation of ***SLC26A7*** in states that are associated with hypokalemia or increased medullary tonicity. Additional studies are needed to ascertain the role of ***SLC26A7*** in enhanced bicarbonate absorption in outer medullary collecting duct in hypokalemia and in acid-base regulation in conditions that are associated with increased medullary tonicity.

ACCESSION NUMBER: 2006170499 MEDLINE <<LOGINID::20080219>>
 DOCUMENT NUMBER: PubMed ID: 16524946
 TITLE: Chloride/bicarbonate exchanger ***SLC26A7*** is localized in endosomes in medullary collecting duct cells and is targeted to the basolateral membrane in hypertonicity and potassium depletion.

AUTHOR: Xu Jie; Worrell Roger T; Li Hong C; Barone Sharon L; Petrovic Snezana; Amlal Hassane; Soleimani Manoocher
 CORPORATE SOURCE: Division of Nephrology and Hypertension, Department of Medicine, University of Cincinnati, 231 Albert Sabin Way,
 MSB 259G, Cincinnati, OH 45267-0585, USA.

CONTRACT NUMBER: DK 62809 (United States NIDDK)
 SOURCE: Journal of the American Society of Nephrology :
 JASN, (2006 Apr) Vol. 17, No. 4, pp. 956-67. Electronic
 Publication: 2006-03-08.
 Journal code: 9013836. ISSN: 1046-6673.

PUB. COUNTRY: United States
 DOCUMENT TYPE: (IN VITRO)

Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200609
 ENTRY DATE: Entered STN: 28 Mar 2006
 Last Updated on STN: 29 Sep 2006
 Entered Medline: 28 Sep 2006

L1 ANSWER 19 OF 134 MEDLINE on STN
 AB Previous studies have indicated that a major fraction of the filtered Cl(-) is reabsorbed via apical membrane Cl(-)/base exchange in the proximal tubule. Recent studies in Slc26a6 null mice have suggested that this transporter mediates only a portion of proximal tubule Cl(-)/base exchange, raising the possibility that one or more unidentified apical membrane transporters may additionally contribute. Recent studies have identified ***Slc26a7*** as another Cl(-)/base exchanger expressed in the kidney. We therefore generated ***Slc26a7*** -specific polyclonal and monoclonal antibodies to examine cellular and subcellular sites of expression in mouse kidney. The specificity of each antibody was verified by immunoblotting and immunofluorescence of COS-7 cells transiently transfected with mouse ***Slc26a7***. Immunofluorescence microscopy of mouse kidney detected the expression of ***Slc26a7*** subapically in proximal tubule cells, and on the basolateral surface of thick ascending limb cells. Similar staining patterns were demonstrated with two antibodies shown to react with different epitopes on ***Slc26a7***. Immunolocalization of ***Slc26a7*** to proximal tubule and thick ascending limb was also observed in rat kidney. We conclude that ***Slc26a7*** is expressed in the proximal tubule and thick ascending limb of the loop of Henle, and it may therefore contribute to anion transport in these nephron segments.

ACCESSION NUMBER: 2006138964 MEDLINE <<LOGINID::20080219>>
 DOCUMENT NUMBER: PubMed ID: 16263805
 TITLE: Immunolocalization of anion transporter ***Slc26a7***

 in mouse kidney.

AUTHOR: Dudas Paul L; Mentone SueAnn; Greineder Colin F;
 Biemesderfer Daniel; Aronson Peter S
 CORPORATE SOURCE: Department of Internal Medicine, Yale University
 School of Medicine, 1 Gilbert St., TAC S-255, P.O. Box 208029,
 New Haven, CT 06520-8029, USA.

CONTRACT NUMBER: 1-F32 DK-067791 (United States NIDDK)
 R01-DK-33793 (United States NIDDK)

SOURCE: American journal of physiology. Renal physiology,
(2006 Apr) Vol. 290, No. 4, pp. F937-45. Electronic
Publication: 2005-11-01.
Journal code: 100901990. ISSN: 0363-6127.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200604
ENTRY DATE: Entered STN: 11 Mar 2006
Last Updated on STN: 14 Apr 2006
Entered Medline: 13 Apr 2006

L1 ANSWER 20 OF 134 MEDLINE on STN
AB BACKGROUND: The anion transporters SLC26A6 (PAT1) and ***SLC26A7

transporting at least chloride, oxalate, sulfate and bicarbonate,
show a
distinct expression and function in different mammalian species.
They are
expressed in kidney, but their exact localization in human kidney
has not
been studied. We therefore examined SLC26A6 and A7 expression in
human
kidneys. METHODS: The localization of SLC26A6 and A7 in different
segments of human nephrons was studied by RT-PCR and
immunohistochemistry
by comparing to the tubular markers PNRA, CD10, Tamm-Horsfall
antigen,
high molecular weight cytokeratin, CK7, AQP2 and H(+)V-ATPase.
RESULTS:
In human kidney, SLC26A6 is expressed in distal segments of
proximal
tubules, parts of the thin and thick ascending limbs of Henle's
loops,
macula densa, distal convoluted tubules and a subpopulation of
intercalated cells of collecting ducts. ***SLC26A7*** is
expressed in
extraglomerular mesangial cells and a subpopulation of intercalated
cells
of collecting ducts. CONCLUSION: Our results show that in human
kidney
SLC26A6 and A7 have a distinct, partially overlapping expression in
distal
segments of nephrons. The distribution partly differs from that
found
previously in rodent kidneys.
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ACCESSION NUMBER: 2005456212 MEDLINE <<LOGINID::20080219>>
DOCUMENT NUMBER: PubMed ID: 15956810
TITLE: SLC26A6 and ***SLC26A7*** anion exchangers have
a
distinct distribution in human kidney.
AUTHOR: Kujala Minna; Tienari Jukka; Lohi Hannes; Elomaa
Outi;
Sariola Hannu; Lehtonen Eero; Kere Juha
CORPORATE SOURCE: Department of Medical Genetics, University of
Helsinki,

SOURCE: Helsinki, Finland.. minna.kujala@helsinki.fi
 Nephron. Experimental nephrology, (2005) Vol. 101,
 No. 2, pp. e50-8. Electronic Publication: 2005-06-14.
 Journal code: 101159770. E-ISSN: 1660-2129.
 PUB. COUNTRY: Switzerland
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, NON-U.S. GOV'T)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200604
 ENTRY DATE: Entered STN: 27 Aug 2005
 Last Updated on STN: 22 Apr 2006
 Entered Medline: 21 Apr 2006

L1 ANSWER 21 OF 134 MEDLINE on STN
 AB Members of the SLC26 transporter family play an essential role in
 several
 epithelial functions, as revealed by diseases associated with
 mutations in
 members of the family. Several members were shown to function as
 Cl(-)
 and HCO(3)(-) transporters that likely play an important role in
 epithelial Cl(-) absorption and HCO(3)(-) secretion. However, the
 mechanism of most transporters is not well understood.
 SLC26A7
 is a member of the SLC26 transporter family reported to be
 expressed in
 the basolateral membrane of the cortical collecting duct and
 parietal
 cells and functions as a coupled Cl(-)/HCO(3)(-) exchanger. In the
 present work we examined the transport properties of ***SLC26A7

 to
 determine its transport characteristics and electrogenicity. We
 found
 that when expressed in Xenopus oocytes or HEK293 cells ***SLC26A7

 functions as a pH(i)-regulated Cl(-) channel with minimal OH(-)/HCO
 (3)(-)
 permeability. Expression of ***SLC26A7*** in oocytes or HEK293
 cells
 generated a Cl(-) current with linear I/V and an instantaneous
 current
 that was voltage- and time-independent. Based on measurement of
 reversal
 potential the selectivity of ***SLC26A7*** is NO(3)(-)>>Cl(-)
 =Br(-)=I(-)
)>SO(4)(2-)=Glu(-), although I(-) partially inhibited the current.
 Incubating the cells with HCO(3)(-) or butyrate acidified the
 cytosol and
 increased the selectivity of ***SLC26A7*** for Cl(-).
 Measurement of
 membrane potential and pH(i) showed minimal OH(-) and HCO(3)(-)
 transport
 by ***SLC26A7*** when the cells were incubated in Cl(-)-
 containing or
 Cl(-)-free media. The activity of ***SLC26A7*** was inhibited
 by all
 inhibitors of anion transporters tested, 4,4'-
 diisothiocyanostilbene-2,2'-
 disulfonic acid, diphenylamine-2-carboxylic acid, and

glybenclamide.

These findings reveal that ***SLC26A7*** functions as a unique Cl(-)

channel that is regulated by intracellular H(+).

ACCESSION NUMBER: 2005091924 MEDLINE <<LOGINID::20080219>>

DOCUMENT NUMBER: PubMed ID: 15591059

TITLE: ***SLC26A7*** is a Cl- channel regulated by intracellular pH.

AUTHOR: Kim Kil Hwan; Shcheynikov Nikolay; Wang Youxue; Muallem

Shmuel

CORPORATE SOURCE: Department of Physiology, University of Texas Southwestern

Medical Center, Dallas, Texas 75390-9040, USA.

SOURCE: The Journal of biological chemistry, (2005 Feb 25) Vol.

280, No. 8, pp. 6463-70. Electronic Publication: 2004-12-09.

Journal code: 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200504

ENTRY DATE: Entered STN: 23 Feb 2005

Last Updated on STN: 9 Apr 2005

Entered Medline: 8 Apr 2005

L1 ANSWER 22 OF 134 MEDLINE on STN

AB ***SLC26A7*** is a recently identified Cl(-)/HCO(3)(-) exchanger that

co-localizes with AE1 on the basolateral membrane of Alpha intercalated

cells (A-IC) in outer medullary collecting duct (OMCD). The purpose of

these studies was to determine whether AE1 and ***SLC26A7*** are

differentially regulated in OMCD in pathophysiologic states.

Toward this

end, the expression and regulation of AE1 and ***SLC26A7*** was examined in water deprivation, a condition known to increase the osmolality of the medulla. Rats were subjected to 3 d of water deprivation while having free access to food. Northern

hybridizations

demonstrated that in the outer medulla, the mRNA expression of

SLC26A7 increased by approximately 300% (P < 0.01 versus control;

n = 3), whereas the expression of AE1 decreased by approximately 50% (P <

0.05 versus control, n = 3) in water-deprived rats. Immunoblot analysis

studies demonstrated that in the outer medulla, ***SLC26A7*** abundance increased by approximately 3.5-fold (P < 0.02 versus

control; n

= 3), whereas the AE1 abundance decreased by approximately 55% (P < 0.05

versus control) in water deprivation. The expression of

SLC26A7

remained unchanged in the kidney cortex and stomach in water

deprivation,

indicating the specificity of ***SLC26A7*** upregulation in

outer medulla. In situ hybridization indicated the exclusive expression of ***SLC26A7*** in the outer medulla and double immunofluorescence labeling confirmed the co-localization of AE1 and ***SLC26A7*** on the basolateral membrane of A-IC cells in OMCD. It is concluded that AE1 and ***SLC26A7*** are differentially regulated in OMCD in water deprivation. On the basis of these results and previous functional studies indicating the activation of ***SLC26A7*** activity by high osmolality, it is proposed that ***SLC26A7*** may play an important role in bicarbonate reabsorption and or cell volume regulation in OMCD (specifically under hypertonic conditions).

ACCESSION NUMBER: 2004380910 MEDLINE <<LOGINID::20080219>>
DOCUMENT NUMBER: PubMed ID: 15284286
TITLE: Differential regulation of basolateral Cl-/HCO3-exchangers
SLC26A7 and AE1 in kidney outer medullary collecting duct.
AUTHOR: Barone Sharon; Amlal Hassane; Xu Jie; Kujala Minna; Kere
CORPORATE SOURCE: Juha; Petrovic Snezana; Soleimani Manoocher
231 Department of Medicine, University of Cincinnati,
Albert Sabin Way, MSB G259, Cincinnati, OH 45267-0585, USA.
CONTRACT NUMBER: DK 54430 (United States NIDDK)
DK 62809 (United States NIDDK)
SOURCE: Journal of the American Society of Nephrology :
JASN, (2004 Aug) Vol. 15, No. 8, pp. 2002-11.
Journal code: 9013836. ISSN: 1046-6673.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200409
ENTRY DATE: Entered STN: 1 Aug 2004
Last Updated on STN: 21 Sep 2004
Entered Medline: 20 Sep 2004

L1 ANSWER 23 OF 134 MEDLINE on STN
AB The outer medullary collecting duct (OMCD) plays an important role in bicarbonate reabsorption and acid-base regulation. An apical V-type H+-ATPase and a basolateral Cl-/HCO3- exchanger, located in intercalated cells of OMCD, mediate the bicarbonate reabsorption. Here we report the identification of a new basolateral Cl-/HCO3- exchanger in OMCD intercalated cells in rat kidney. Northern hybridizations

demonstrated the predominant expression of this transporter, also known as ***SLC26A7***, in the outer medulla, with lower expression levels in the inner medulla. ***SLC26A7*** was recognized as a approximately 90-kDa band in the outer medulla by immunoblot analysis and was localized on the basolateral membrane of a subset of OMCD cells by immunocytochemical staining. No labeling was detected in the cortex. Double-immunofluorescence labeling with the aquaporin-2 and ***SLC26A7*** antibodies or anion exchanger-1 and ***SLC26A7*** antibodies identified the ***SLC26A7***-expressing cells as alpha-intercalated cells. Functional studies in oocytes demonstrated that increasing the osmolality of the media (to simulate the physiological milieu in the medulla) increased the Cl⁻/HCO₃⁻ exchanger activity mediated via ***SLC26A7*** by about threefold (P < 0.02 vs. normal condition). We propose that ***SLC26A7*** is a basolateral Cl⁻/HCO₃⁻ exchanger in intercalated cells of the OMCD and may play an important role in bicarbonate reabsorption in medullary collecting duct.

ACCESSION NUMBER: 2003576247 MEDLINE <<LOGINID::20080219>>
DOCUMENT NUMBER: PubMed ID: 12965893
TITLE: ***SLC26A7*** : a basolateral Cl⁻/HCO₃⁻ exchanger specific to intercalated cells of the outer medullary collecting duct.
AUTHOR: Petrovic Snezana; Barone Sharon; Xu Jie; Conforti Laura; Ma Liyun; Kujala Minna; Kere Juha; Soleimani Manoocher
CORPORATE SOURCE: Division of Nephrology and Hypertension, Department of Medicine, University of Cincinnati, 231 Albert Sabin Way, MSB G259, Cincinnati, OH 45267-0585, USA.
CONTRACT NUMBER: DK 54430 (United States NIDDK)
SOURCE: American journal of physiology. Renal physiology, (2004 Jan) Vol. 286, No. 1, pp. F161-9. Electronic
Publication: 2003-09-09.
JOURNAL CODE: 100901990. ISSN: 0363-6127.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200402
ENTRY DATE: Entered STN: 16 Dec 2003
Last Updated on STN: 11 Feb 2004
Entered Medline: 10 Feb 2004

L1 ANSWER 24 OF 134 MEDLINE on STN
AB The basolateral Cl(-)/HCO(3)(-) exchanger in parietal cells plays

an essential role in gastric acid secretion mediated via the apical gastric H(+)-K(+)-ATPase. Here, we report the identification of a new Cl(-)/HCO(3)(-) exchanger, which shows exclusive expression in mouse stomach and kidney, with expression in the stomach limited to the basolateral membrane of gastric parietal cells. Tissue distribution studies by RT-PCR and Northern hybridizations demonstrated the exclusive expression of this transporter, also known as ***SLC26A7***, to stomach and kidney, with the stomach expression significantly more abundant. No expression was detected in the intestine. Cellular distribution studies by RT-PCR and Northern hybridizations demonstrated predominant localization of ***SLC26A7*** in gastric parietal cells. Immunofluorescence labeling localized this exchanger exclusively to the basolateral membrane of gastric parietal cells, and functional studies in oocytes indicated that ***SLC26A7*** is a DIDS-sensitive Cl(-)/HCO(3)(-) exchanger that is active in both acidic and alkaline pH(i). On the basis of its unique expression pattern and function, we propose that ***SLC26A7*** is a basolateral Cl(-)/HCO(3)(-) exchanger in gastric parietal cells and plays a major role in gastric acid secretion.

ACCESSION NUMBER: 2003250506 MEDLINE <<LOGINID::20080219>>
DOCUMENT NUMBER: PubMed ID: 12736153
TITLE: Identification of a basolateral Cl-/HCO3- exchanger specific to gastric parietal cells.
AUTHOR: Petrovic Snezana; Ju Xie; Barone Sharon; Seidler Ursula;
Manoocher Alper Seth L; Lohi Hannes; Kere Juha; Soleimani
CORPORATE SOURCE: Department of Medicine, University of Cincinnati, Cincinnati 45267, USA.
CONTRACT NUMBER: DK-52820 (United States NIDDK)
DK-54430 (United States NIDDK)
SOURCE: American journal of physiology. Gastrointestinal and liver physiology, (2003 Jun) Vol. 284, No. 6, pp. G1093-103.
Journal code: 100901227. ISSN: 0193-1857.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200306
ENTRY DATE: Entered STN: 31 May 2003
Last Updated on STN: 24 Jun 2003
Entered Medline: 23 Jun 2003

AB A second distinct family of anion exchangers, SLC26, in addition to the classical SLC4 (or anion exchanger) family, has recently been delineated.

Particular interest in this gene family is stimulated by the fact that the

SLC26A2, SLC26A3, and SLC26A4 genes have been recognized as the disease

genes mutated in diastrophic dysplasia, congenital chloride diarrhea, and

Pendred syndrome, respectively. We report the expansion of the SLC26 gene

family by characterizing three novel tissue-specific members, named ***SLC26A7***, SLC26A8, and SLC26A9, on chromosomes 8, 6, and

1, respectively. The ***SLC26A7*** -A9 proteins are structurally very

similar at the amino acid level to the previous family members and show

tissue-specific expression in kidney, testis, and lung, respectively.

More detailed characterization by immunohistochemistry and/or in situ

hybridization localized ***SLC26A7*** to distal segments of nephrons,

SLC26A8 to developing spermatocytes, and SLC26A9 to the luminal side of

the bronchiolar and alveolar epithelium of lung. Expression of ***SLC26A7*** -A9 proteins in Xenopus oocytes demonstrated

chloride, sulfate, and oxalate transport activity, suggesting that they encode

functional anion exchangers. The functional characterization of the novel

tissue-specific members may provide new insights to anion transport physiology in different parts of body.

ACCESSION NUMBER: 2002234636 MEDLINE <<LOGINID::20080219>>

DOCUMENT NUMBER: PubMed ID: 11834742

TITLE: Functional characterization of three novel tissue-specific

anion exchangers ***SLC26A7***, -A8, and -A9.

AUTHOR: Lohi Hannes; Kujala Minna; Makela Siru; Lehtonen Eero;

Kestila Marjo; Saarialho-Kere Ulpu; Markovich

Daniel; Kere

Juha

CORPORATE SOURCE: Department of Medical Genetics, Biomedicum Helsinki and

Helsinki University Central Hospital, P. O. Box 63 (Haartmaninkatu 8), 00014 University of Helsinki,

Helsinki,

Finland.

SOURCE: The Journal of biological chemistry, (2002 Apr 19) Vol.

277, No. 16, pp. 14246-54. Electronic Publication: 2002-02-07.

Journal code: 2985121R. ISSN: 0021-9258.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)

LANGUAGE: English

FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF331521; GENBANK-AF331522; GENBANK-AF331525
ENTRY MONTH: 200206
ENTRY DATE: Entered STN: 26 Apr 2002
Last Updated on STN: 5 Jan 2003
Entered Medline: 7 Jun 2002

L1 ANSWER 26 OF 134 MEDLINE on STN
AB A unique characteristic of endothelial cells from high endothelial
venules
(HEVEC) in lymphoid organs and chronically inflamed tissues is
their
capacity to incorporate large amounts of sulfate into sialomucin-
type
counter-receptors for the lymphocyte homing receptor L-selectin.
We have
previously shown that HEVEC express two functional classes of
sulfate
transporters: sodium/sulfate cotransporters and sulfate/anion
exchangers.
Here, we report the molecular cloning from human HEVEC of a 2.9-kb
cDNA
encoding ***SLC26A7*** , a novel member of the SLC26 (solute
carrier
26) sulfate/anion exchanger family. ***SLC26A7*** exhibits 30%
identity with three known sulfate transporters from the SLC26
family:
SLC26A2 (also known as DTDST), SLC26A1 (also known as SAT1), and
SLC26A3
(also known as DRA). Northern blot analysis revealed specific
expression
of ***SLC26A7*** mRNA in kidney. Alternative splicing and
polyadenylation of ***SLC26A7*** pre-mRNA in kidney suggest the
existence of two protein isoforms, ***SLC26A7*** .1 and
SLC26A7
.2, differing in their carboxy termini.
ACCESSION NUMBER: 2002099720 MEDLINE <<LOGINID::20080219>>
DOCUMENT NUMBER: PubMed ID: 11829495
TITLE: Molecular cloning of ***SLC26A7*** , a novel
member of
the SLC26 sulfate/anion transporter family, from
high
endothelial venules and kidney.
AUTHOR: Vincourt Jean-Baptiste; Jullien Denis; Kossida
Sophia;
Amalric Francois; Girard Jean-Philippe
CORPORATE SOURCE: Laboratoire de Biologie Vasculaire, Institut de
Pharmacologie et de Biologie Structurale du CNRS,
205 route
de Narbonne, Toulouse, 31077, France.
SOURCE: Genomics, (2002 Feb) Vol. 79, No. 2, pp. 249-56.
Journal code: 8800135. ISSN: 0888-7543.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AJ413228; GENBANK-AJ413229; GENBANK-AJ413230
ENTRY MONTH: 200209
ENTRY DATE: Entered STN: 7 Feb 2002
Last Updated on STN: 18 Sep 2002

Entered Medline: 17 Sep 2002

L1 ANSWER 27 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
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STN

AB To date three potential candidates for parietal cell basolateral
Cl- entry
have been described: the highly 4,4'-diisothiocyanatostilbene-2,2'-
disulfonic acid (DIDS)-sensitive Cl-/HCO₃ exchanger AE2, the HCO₃-
and
lowly DIDS-sensitive ***SLC26A7*** protein, and the Na+-2Cl(-)K
(+)
cotransporter (NKCC1). In this study we investigate the
contribution of
these pathways to secretagogue stimulated acid secretion.
Individually
hand-dissected rat gastric glands were microfluorimetrically
monitored for
Cl- influx and pH(i) changes. Transporter activity was determined
by
varying ion content and through the use of pharmacological
inhibitors.
Expression of ***SLC26A7*** in rat parietal cells was shown by
immunohistochemistry and Western blot. ***SLC26A7*** was
inhibited by
5-Nitro-2-(3-phenylpropyl-amino)benzoic acid (NPPB) (100 μ M) in
the
Xenopus laevis oocyte expression system. Cl- influx in parietal
cells was
enhanced by histamine, depended partially on endogenous HCO₃-
synthesis
and completely on extracellular Na+. Removal and subsequent
readdition of
Cl- revealed a low and a high DIDS-sensitive HCO₃- extrusion system
contributing to Cl- uptake. At acidic pH(i), however, H+ extrusion
via
the H+,K+-ATPase depending on Cl- uptake was abolished only in the
presence of 100 μ M (NPPB) and at high (250 μ M) DIDS
concentration.
There was no effect of the NKCC inhibitor bumetanide on stimulated
H+
extrusion. These results would be compatible with ***SLC26A7***
as a
Cl- uptake system under histamine stimulation.

ACCESSION NUMBER: 2007:569908 BIOSIS <<LOGINID::20080219>>

DOCUMENT NUMBER: PREV200700571792

TITLE: ***SLC26A7*** Can function as a chloride-loading
mechanism in parietal cells.

AUTHOR(S): Kosiek, Ortrud; Busque, Stephanie M.; Foeller,
Michael;

Shcheynikov, Nikolay; Kirchhoff, Philipp; Bleich,
Markus;

Muallem, Shmuel; Geibel, John P. [Reprint Author]
CORPORATE SOURCE: Yale Univ, Sch Med, Dept Surg, BML 265,310 Cedar St,
New

Haven, CT 06520 USA
john.geibel@yale.edu

SOURCE: Pfluegers Archiv European Journal of Physiology,
(SEP 2007)

Vol. 454, No. 6, pp. 989-998.
CODEN: PFLABK. ISSN: 0031-6768.

DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Nov 2007
Last Updated on STN: 7 Nov 2007

L1 ANSWER 28 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
Corporation on
STN

AB Uptake of SO42- by articular chondrocytes is an essential step in
the
pathway for sulphation of glycosammoglycans (GAGs), with mutations
in
So(4)(2-) transport proteins resulting in abnormalities of skeletal
growth. In the present study, the transporters mediating SO42-
transport
in bovine articular chondrocytes have been characterized.
Expression of
candidate transporters was determined using RT-PCR, while SO42-
transport
was measured in radioisotope flux experiments. RT-PCR experiments
showed
that bovine articular chondrocytes express three transporters known
to
transport SO42- : AE2 (SLC4a2), DTDST (SLC26a2), and SLC26a11.
Other
transporters-NaS-1 (SLC13a1), SAT-1 (SLC26a1), DRA (SLC26a3),
SLC26a6
(PAT1), ***SLC26a7*** , SLC26a8 (Tat-1), and SLC26a9-were,
however, not
detected. In functional experiments, SO42- uptake was temperature
sensitive, inhibited by 60% by DIDS (50 mu m) and exhibited
saturation
kinetics, with a K-m value of 16 mM. Uptake was also inhibited at
alkaline extracellular pH. In further experiments, a K-i value for
DIDS
inhibition of SO42- efflux of 5 mu M was recorded. A DIDS-
sensitive
component of SO42- efflux persisted in solutions lacking Cl- ions.
These
data are interpreted as evidence for the preferential operation of
carrier-mediated exchange of SO42- - for Cl-, while an alternative
SO42-
-OH- exchange mode is also possible. (c) 2007 Orthopaedic Research
Society.

ACCESSION NUMBER: 2007:561824 BIOSIS <<LOGINID::20080219>>

DOCUMENT NUMBER: PREV200700563206

TITLE: Characterization of sulphate transporters in
isolated

bovine articular chondrocytes.
AUTHOR(S): Meredith, David; Gehl, Katharina A.; Seymour, John;
Ellory,

J. Clive; Wilkins, Robert J. [Reprint Author]
CORPORATE SOURCE: Dept Physiol Anat and Genet, Sherrington Bldg, Pk Rd,
Oxford

OX1 3PT, UK
robert.wilkins@physiol.ox.ac.uk
SOURCE: Journal of Orthopaedic Research, (SEP 2007) Vol. 25,
No. 9,

pp. 1145-1153.
CODEN: JOREDR. ISSN: 0736-0266.
DOCUMENT TYPE: Article

LANGUAGE: English
ENTRY DATE: Entered STN: 31 Oct 2007
Last Updated on STN: 21 Nov 2007

L1 ANSWER 29 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AB Aims: Anions have an important role in the regulation of airway surface

liquid (ASL) volume, viscosity and pH. However, functional localization

and regulation of anion exchangers (AEs) have not been clearly described.

The aim of this study was to investigate the regulation of AE mRNA expression level in accordance with mucociliary differentiation and the

functional expression of AEs cultured normal human nasal epithelial (NHNE)

cells. Methods: Nasal mucosal specimens from three patients are obtained

and serially cultured cells are subjected to morphological examinations,

RT-PCR, Western blot analysis and immunocytochemistry. AE activity is

assessed by pH_i measurements. Results: Expression of ciliated cells on the

apical membrane and expression of MUC5AC, a marker of mucous differentiation, increased with time. AE2 and SLC26A4 mRNA

expression

decreased as mucociliary differentiation progressed, and AE4, ***SLC26A7*** and SLC26A8 mRNA expression increased on the 14th and 28th

day after confluence. Accordingly, AE4 protein expression also progressively increased. AE activity in 100 mM K⁺ buffer solutions was

nearly twofold higher than that in 5 mM K⁺ buffer solutions.

Moreover,

only luminal AE activity increased about fourfold over the control in the

presence of 5 μM forskolin. In the presence of 100 μM adenosine-5

'-triphosphate (ATP) which evokes intracellular calcium signalling through

activation of purinergic receptors, only luminal AE activity was again

significantly increased. On the other hand, 500 μM 4,4

'-diisothiocyanostilbene-2,2 '-disulfonic acid (DIDS), an inhibitor of

most SLC4 and SLC26AE isoforms, nearly abolished AE activity in both

luminal and basolateral membranes. We found that AE activity was affected

by intracellular cAMP and calcium signalling in the luminal membrane and

was DIDS-sensitive in both membranes of cultured NHNE

cells. Conclusion:

Our findings through molecular and functional studies using cultured NHNE

cells suggest that AEs may have an important role in the regulation of

ASL.

ACCESSION NUMBER: 2007:547307 BIOSIS <<LOGINID::20080219>>
 DOCUMENT NUMBER: PREV200700529634
 TITLE: Molecular and functional expression of anion
 exchangers in cultured normal human nasal epithelial cells.
 AUTHOR(S): Shin, J.-H.; Son, E. J.; Lee, H. S.; Kim, S. J.;
 Kim, K.; Choi, J. Y.; Lee, M. G.; Yoon, J.-H. [Reprint
 Author]
 CORPORATE SOURCE: Yonsei Univ, Coll Med, Dept Otorhinolaryngol, 134
 Shinchon-dong, Seoul 120752, South Korea
 jhyoon@yumc.yonsei.ac.kr
 SOURCE: Acta Physiologica, (OCT 2007) Vol. 191, No. 2, pp.
 99-110.
 ISSN: 1748-1708.
 DOCUMENT TYPE: Article
 LANGUAGE: English
 ENTRY DATE: Entered STN: 17 Oct 2007
 Last Updated on STN: 17 Oct 2007

L1 ANSWER 30 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
 Corporation on

STN
 AB Appropriate intraluminal microenvironment in the epididymis is
 essential
 for maturation of sperm. To clarify whether the anion transporters
 SLC26A2, SLC26A6, ***SLC26A7***, and SLC26A8 might participate
 in
 generating this proper intraluminal milieu, we studied the
 localization of
 these proteins in the human efferent and the epididymal ducts by
 immunohistochemistry. In addition, immunohistochemistry of several
 SLC26-interacting proteins was performed: the Na⁺/H⁺ exchanger 3
 (NHE3),
 the Cl⁻ channel cystic fibrosis transmembrane conductance regulator
 (CFTR), the proton pump V-ATPase, their regulator Na⁺/H⁺ exchanger
 regulating factor 1 (NHERF-1), and carbonic anhydrase II (CAII).
 Our
 results show that SLC26A6, CFTR, NHE3, and NHERF-1 are co-expressed
 on the
 apical side of the nonciliated cells, and SLC26A2 appears in the
 cilia of
 the ciliated cells in the human efferent ducts. In the epididymal
 ducts,
 SLC26A6, CFTR, NHERF-1, CAII, and V-ATPase (B and E subunits) were
 co-localized to the apical mitochondria rich cells, while
 SLC26A7
 was expressed in a subgroup of basal cells. SLC26A8 was not found
 in the
 structures studied. This is the first study describing the
 focalization
 of SLC26A2, A6 and A7, and NHERF-1 in the efferent and the
 epididymal
 ducts. Immunolocalization of human CFTR, NHE3, CAII, and V-ATPase
 in
 these structures differs partly from previous reports from rodents.
 Our
 findings suggest roles for these proteins in male fertility, either
 independently or through interaction and reciprocal regulation with
 co-localized proteins shown to affect fertility, when disrupted.

ACCESSION NUMBER: 2007:421821 BIOSIS <<LOGINID::20080219>>

DOCUMENT NUMBER: PREV200700426369
 TITLE: Expression of ion transport-associated proteins in human efferent and epididymal ducts.
 AUTHOR(S): Kujala, Minna [Reprint Author]; Hihnala, Satu; Tienari, Jukka; Kaunisto, Kari; Hastbacka, Johanna; Holmberg, Christer; Kere, Juha; Hoglund, Pia
 CORPORATE SOURCE: Univ Helsinki, Dept Med Genet, POB 63, FIN-00014 Helsinki, Finland
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 SOURCE: minna.kujala@helsinki.fi
 Reproduction (Cambridge), (APR 2007) Vol. 133, No. 4, pp. 775-784.
 ISSN: 1470-1626.
 DOCUMENT TYPE: Article
 LANGUAGE: English
 ENTRY DATE: Entered STN: 8 Aug 2007
 Last Updated on STN: 22 Aug 2007

L1 ANSWER 31 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AB Human red cell anion exchanger AE1 (band 3) is an electroneutral Cl-HCO₃- exchanger with 12-14 transmembrane spans (TMs). Previous work using *Xenopus* oocytes has shown that two co-expressed fragments of AE1 lacking TMs 6 and 7 are capable of forming a stilbene disulphonate-sensitive 16 Cl-influx pathway, reminiscent of intact AE1. In the present study, we create a single construct, AE1 Delta(6:7), representing the intact protein lacking TMs 6 and 7. We expressed this construct in *Xenopus* oocytes and evaluated it employing a combination of two-electrode voltage clamp and pH-sensitive microelectrodes. We found that, whereas AE1 Delta (6:7) has some electroneutral Cl-base exchange activity, the protein also forms a novel anion-conductive pathway that is blocked by DIDS. The mutation Ly(S539)Ala at the covalent DIDS-reaction site of AE1 reduced the DIDS sensitivity, demonstrating that (1) the conductive pathway is intrinsic to AE1 Delta(6: 7) and (2) the conductive pathway has some commonality with the electroneutral anion-exchange pathway. The conductance has an anion-permeability sequence: NO₃-approximate to I- > NO₂- > Br- cl > so(4) (2-) approximate to HCO₃ (-) approximate to gluconate(-) approximate to aspartate(-) approximate to cyclamate(-). It may also have a limited permeability to Na⁺ and the zwitterion taurine. Although this conductive

pathway is not a usual feature of intact mammalian AE1, it shares many properties with the anion-conductive pathways intrinsic to two other Cl-HCO₃- exchangers, trout AE1 and mammalian ***SLC26A7*** .

ACCESSION NUMBER: 2007:396873 BIOSIS <<LOGINID::20080219>>
DOCUMENT NUMBER: PREV200700395772
TITLE: A conductive pathway generated from fragments of the human red cell anion exchanger AE1.
AUTHOR(S): Parker, Mark D. [Reprint Author]; Young, Mark T.; Daly, Christopher M.; Meech, Robert W.; Boron, Walter F.; Tanner, Michael J. A.
CORPORATE SOURCE: Yale Univ, Sch Med, Dept Cellular and Mol Physiol, 333 Cedar St, SHM B-127, New Haven, CT 06510 USA
mark.parker@yale.edu
SOURCE: Journal of Physiology (Oxford), (MAY 15 2007) Vol. 581, No. 1, pp. 33-50.
CODEN: JPHYA7. ISSN: 0022-3751.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 18 Jul 2007
Last Updated on STN: 18 Jul 2007

L1 ANSWER 32 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AB Sulfate is essential for normal cellular function. The kidney plays a major role in sulfate homeostasis. Sulfate is freely filtered and then undergoes net reabsorption in the proximal tubule. The apical membrane Na⁺/sulfate cotransporter NaS1 (SLC13A1) has a major role in mediating proximal tubule sulfate reabsorption, as demonstrated by the findings of hyposulfatemia and hypersulfaturia in NaS1-null mice. The anion exchanger SAT 1 (SLC26A1), the founding member of the SLC26 sulfate transporter family, mediates sulfate exit across the basolateral membrane to complete the process of transtubular sulfate reabsorption. Another member of this family, CFEX (SLC26A6), is present at the apical membrane of proximal tubular cells. It also can transport sulfate by anion exchange, which probably mediates backflux of sulfate into the lumen. Knockout mouse studies have demonstrated a major role of CFEX as an apical membrane Cl⁻/oxalate exchanger that contributes to NaCl reabsorption in the proximal tubule. Several additional SLC26 family members mediate sulfate transport and show some level of renal expression (e.g., SLC26A2,

SLC26A7 , SLC26A11). Their roles in mediating renal tubular sulfate transport are presently unknown. This paper reviews current data available on the function and regulation of three sulfate transporters (NaSl, SAT1, and CFEX) and their physiological roles in the kidney.

ACCESSION NUMBER: 2007:315555 BIOSIS <<LOGINID::20080219>>
DOCUMENT NUMBER: PREV200700315822
TITLE: Specificity and regulation of renal sulfate transporters.
AUTHOR(S): Markovich, Daniel [Reprint Author]; Aronson, Peter S.
CORPORATE SOURCE: Univ Queensland, Sch Biomed Sci, Dept Physiol and Pharmacol, Brisbane, Qld 4072, Australia
d.markovich@uq.edu.au; peter.aronson@yale.edu
SOURCE: Annual Review of Physiology, (2007) Vol. 69, pp. 361-375.
CODEN: ARPHAD. ISSN: 0066-4278.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 24 May 2007
Last Updated on STN: 24 May 2007

L1 ANSWER 33 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AB ***SLC26A7*** is a Cl⁻/HCO₃⁻ exchanger that is expressed on the basolateral membrane and in the cytoplasm of two distinct acid-secreting epithelial cells: The A-intercalated cells in the kidney outer medullary collecting duct and the gastric parietal cells. The intracellular localization of ***SLC26A7*** suggests the possibility of trafficking between cell membrane and intracellular compartments. For testing this hypothesis, full-length human ***SLC26A7*** cDNA was fused with green fluorescence protein and transiently expressed in MDCK epithelial cells. In monolayer cells in isotonic medium, ***SLC26A7*** showed punctate distribution throughout the cytoplasm. However, in medium that was made hypertonic for 16 h, ***SLC26A7*** was detected predominantly in the plasma membrane. The presence of mitogen-activated protein kinase inhibitors blocked the trafficking of ***SLC26A7*** to the plasma membrane. Double-labeling studies demonstrated the localization of ***SLC26A7*** to the transferrin receptor-positive endosomes.

A chimera that was composed of the amino terminal fragment of ***SLC26A7*** and the carboxyl terminal fragment of SLC26A1, and a C-terminal-truncated ***SLC26A7*** were retained in the cytoplasm in hypertonicity. In separate studies, ***SLC26A7*** showed predominant localization

in plasma membrane in potassium-depleted isotonic medium (0.5 or 2 mEq/L KCl) versus cytoplasmic distribution in normal potassium isotonic medium (4 mEq/L). It is concluded that ***SLC26A7*** is present in endosomes, and its targeting to the basolateral membrane is increased in hypertonicity and potassium depletion. The trafficking to the cell surface suggests novel functional upregulation of ***SLC26A7*** in

states that are associated with hypokalemia or increased medullary tonicity. Additional studies are needed to ascertain the role of ***SLC26A7*** in enhanced bicarbonate absorption in outer medullary collecting duct in hypokalemia and in acid-base regulation in conditions

that are associated with increased medullary tonicity.

ACCESSION NUMBER: 2007:141761 BIOSIS <<LOGINID::20080219>>

DOCUMENT NUMBER: PREV200700145629

TITLE: Chloride/bicarbonate exchanger ***SLC26A7*** is localized in endosomes in medullary collecting duct cells

and is targeted to the basolateral membrane in hypertonicity and potassium depletion.

AUTHOR(S): Xu, Jie; Worrell, Roger T.; Li, Hong C.; Barone, Sharon L.;

Petrovic, Snezana; Amlal, Hassane; Soleimani,

Manoocher

[Reprint Author]

CORPORATE SOURCE: Univ Cincinnati, Dept Med, Div Nephrol and Hypertens, 231

Albert Sabin Way, MSB 259G, Cincinnati, OH 45267 USA
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SOURCE: Journal of the American Society of Nephrology, (APR 2006)

Vol. 17, No. 4, pp. 956-967.

CODEN: JASNEU. ISSN: 1046-6673.

DOCUMENT TYPE: Article

LANGUAGE: English

OTHER SOURCE: GenBank-AF349043; EMBL-AF349043; DDBJ-AF349043; GenBank-NM052832; EMBL-NM052832; DDBJ-NM052832

ENTRY DATE: Entered STN: 28 Feb 2007

Last Updated on STN: 28 Feb 2007

L1 ANSWER 34 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on

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AB ***SLC26A7*** is a newly identified basolateral Cl-/HCO3-exchanger

specific to alpha-intercalated cells of the outer medullary collecting duct (OMCD). The purpose of the present experiments was to examine the

expression of ***SLC26A7*** in kidneys of vasopressin-deficient Brattleboro rats before and after treatment with desamino-Cys(1),D-Arg(8)-

vasopressin (dDAVP). Brattleboro rats were treated with dDAVP, a vasopressin analog, for 8 days, and their kidneys were examined for the

expression of ***SLC26A7***. The expression of ***SLC26A7***

protein, as examined by immunofluorescence, was undetectable in kidneys of Brattleboro rats. However, treatment with dDAVP induced expression of

SLC26A7 protein, restoring it to levels observed in normal rats.

These results were verified by Western blot analysis. The mRNA expression

of ***SLC26A7*** remained unchanged in response to dDAVP.

Immunofluorescent labeling demonstrated abundant levels of anion exchanger

type 1 in the OMCD of Brattleboro rats and a mild reduction in response to

dDAVP. The abundance of H⁺-ATPase was not affected by dDAVP. The increased ***SLC26A7*** expression directly correlated with enhanced

aquaporin-2 expression, which is proportional to increased interstitial

osmolarity in the medulla. In conclusion, vasopressin increases the

expression of ***SLC26A7*** protein through posttranscriptional mechanisms in the OMCD. The induction of ***SLC26A7*** by vasopressin

in OMCD cells of Brattleboro rats is likely an attempt by cells to regulate their cell volume and maintain HCO₃⁻ absorption in a state associated with increased interstitial medullary tonicity.

ACCESSION NUMBER: 2006:359507 BIOSIS <<LOGINID::20080219>>

DOCUMENT NUMBER: PREV200600351918

TITLE: Vasopressin induces expression of the Cl⁻/HCO₃⁻ exchanger

SLC26A7 in kidney medullary collecting ducts of

Brattleboro rats.

AUTHOR(S): Petrovic, Snezana [Reprint Author]; Amlal, Hassane; Sun,

Xuming; Karet, Fiona; Barone, Sharon; Soleimani,

Manoocher

CORPORATE SOURCE: Univ Cincinnati, Sch Med, Dept Med, Div Nephrol and Hypertens, 231 Albert Sabin Way, MSB 259G, Cincinnati, OH

45267 USA

Snezana.Petrovic@uc.edu; Manoocher.Soleimani@uc.edu

SOURCE: American Journal of Physiology - Renal Physiology, (MAY

2006) Vol. 290, No. 5, pp. F1194-F1201.

ISSN: 0363-6127.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 19 Jul 2006

Last Updated on STN: 19 Jul 2006

L1 ANSWER 35 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AB Some Slc26 family members function as Cl⁻/HCO₃⁻ exchangers

(Slc26a3, -a4, -a6), whereas others function as Cl⁻ channels (***Slc26a7***)

or

anion-gated molecular motors (Slc26a5). We have reported that Slc26a9

functions as a Cl⁻/HCO₃⁻ exchanger with a high Cl⁻ conductance. We

have now evaluated the role of Sulfate Transporter Anti-Sigma factor antagonist (STAS) domain, located at the protein's C-terminus, by deleting the STAS domain (Slc26a9-Delta STAS). We expressed mouse Slc26a9 and Slc26a9-Delta STAS in *Xenopus* oocytes and measured membrane potential (V-m), currents, and intracellular pH and Cl⁻ using microelectrodes. Slc26a9-Delta STAS deletes the STAS domain leaving intact the final C-terminus. This deletion reduced Slc26a9 dependent currents. However, Slc26a9-Delta STAS was still capable of HCO₃⁻ transport showing primarily a reduction in Cl⁻ conductance. To localize Slc26a9 protein we developed a chicken antibody against a C-terminal peptide of mouse Slc26a9. Slc26a9 is found in rat respiratory tract (tracheal submucosal glands) and gut (esophagus and stomach). Our antibody not only recognized rat but also human SLC26A9 in Calu-3 cells (human airway submucosal gland cell-line). We conclude that Slc26a9 is a Cl⁻/HCO₃⁻ exchanger, and, like ***Slc26a7***, shows Cl⁻ channel-like currents which are diminished by the deletion of the STAS domain. Regulation of Slc26a9-dependent Cl⁻ conductance could be important for anion transport in the respiratory and gastrointestinal systems.

ACCESSION NUMBER: 2006:337390 BIOSIS <<LOGINID::20080219>>
DOCUMENT NUMBER: PREV200600335949
TITLE: Localization of Slc26a9 and role of the STAS domain.
AUTHOR(S): Sindic, Aleksandra [Reprint Author]; Plata, Consuelo; Sussman, Caroline R.; Mount, David B.; Chang, Min-Hwang; Romero, Michael F.
CORPORATE SOURCE: Case Western Reserve Univ, Sch Med, Cleveland, OH 44104 USA
SOURCE: FASEB Journal, (MAR 7 2006) Vol. 20, No. 5, Part 2, pp. A839.
Meeting Info.: Experimental Biology 2006 Meeting. San Francisco, CA, USA. April 01 -05, 2006. Amer Assoc Anatomists; Amer Physiol Soc; Amer Soc Biochem & Mol Biol; Amer Soc Investigat Pathol; Amer Soc Nutr; Amer Soc Pharmacol & Expt Therapeut.
CODEN: FAJOEC. ISSN: 0892-6638.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Jul 2006
Last Updated on STN: 5 Jul 2006

L1 ANSWER 36 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
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AB Previous studies have indicated that a major fraction of the
filtered Cl-
is reabsorbed via apical membrane Cl-/base exchange in the proximal
tubule. Recent studies in Slc26a6 null mice have suggested that
this
transporter mediates only a portion of proximal tubule Cl-/base
exchange,
raising the possibility that one or more unidentified apical
membrane
transporters may additionally contribute. Recent studies have
identified
Slc26a7 as another Cl-/base exchanger expressed in the
kidney. We
therefore generated ***Slc26a7*** -specific polyclonal and
monoclonal
antibodies to examine cellular and subcellular sites of expression
in
mouse kidney. The specificity of each antibody was verified by
immunoblotting and immunofluorescence of COS-7 cells transiently
transfected with mouse ***Slc26a7***. Immunofluorescence
microscopy
of mouse kidney detected the expression of ***Slc26a7***
subapically
in proximal tubule cells, and on the basolateral surface of thick
ascending limb cells. Similar staining patterns were demonstrated
with
two antibodies shown to react with different epitopes on
Slc26a7
. Immunolocalization of ***Slc26a7*** to proximal tubule and
thick
ascending limb was also observed in rat kidney. We conclude that
Slc26a7 is expressed in the proximal tubule and thick
ascending
limb of the loop of Henle, and it may therefore contribute to anion
transport in these nephron segments.
ACCESSION NUMBER: 2006:297360 BIOSIS <<LOGINID::20080219>>
DOCUMENT NUMBER: PREV200600288999
TITLE: Immunolocalization of anion transporter ***Slc26a7

in mouse kidney.
AUTHOR(S): Dudas, Paul L.; Mentone, SueAnn; Greineder, Colin
F.;
Biemesderfer, Daniel; Aronson, Peter S. [Reprint
Author]
CORPORATE SOURCE: Yale Univ, Sch Med, Dept Internal Med, 1 Gilbert
St, TAC
S-255, POB 208029, New Haven, CT 06520 USA
peter.aronson@yale.edu
SOURCE: American Journal of Physiology - Renal Physiology,
(APR
2006) Vol. 290, No. 4, pp. F937-F945.
ISSN: 0363-6127.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 31 May 2006
Last Updated on STN: 31 May 2006

L1 ANSWER 37 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson

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STN
AB Background: The anion transporters SLC26A6 (PAT1) and ***SLC26A7

,
transporting at least chloride, oxalate, sulfate and bicarbonate,
show a
distinct expression and function in different mammalian species.
They are
expressed in kidney, but their exact localization in human kidney
has not
been studied. We therefore examined SLC26A6 and A7 expression in
human
kidneys. Methods: The localization of SLC26A6 and A7 in different
segments
of human nephrons was studied by RT-PCR and immunohistochemistry by
comparing to the tubular markers PNRA, CD10, Tamm-Horsfall antigen,
high
molecular weight cytokeratin, CK7, AQP2 and H+V-ATPase. Results:
In human
kidney, SLC26A6 is expressed in distal segments of proximal
tubules, parts
of the thin and thick ascending limbs of Henle's loops, macula
densa,
distal convoluted tubules and a subpopulation of intercalated cells
of
collecting ducts. ***SLC26A7*** is expressed in
extraglomerular
mesangial cells and a subpopulation of intercalated cells of
collecting
ducts. Conclusion: Our results show that in human kidney SLC26A6
and A7
have a distinct, partially overlapping expression in distal
segments of
nephrons. The distribution partly differs from that found
previously in
rodent kidneys. Copyright (C) 2005 S. Karger AG, Basel.
ACCESSION NUMBER: 2005:517343 BIOSIS <<LOGINID::20080219>>
DOCUMENT NUMBER: PREV200510307853
TITLE: SLC26A6 and ***SLC26A7*** anion exchangers have
a
distinct distribution in human kidney.
AUTHOR(S): Kujala, Minna [Reprint Author]; Tienari, Jukka;
Lohi,
Hannes; Elomaa, Outi; Sariola, Hannu; Lehtonen,
Eero; Kere,
Juha
CORPORATE SOURCE: Univ Helsinki, Biomedicum Helsinki, Dept Med Genet,
POB 63,
FI-00014 Helsinki, Finland
minna.kujala@helsinki.fi
SOURCE: Nephron Experimental Nephrology, (2005) Vol. 101,
No. 2,
pp. E50-E58.
ISSN: 1660-2129.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Nov 2005
Last Updated on STN: 23 Nov 2005

STN

AB Members of the SLC26 transporter family play an essential role in several epithelial functions, as revealed by diseases associated with mutations in members of the family. Several members were shown to function as Cl⁻ and HCO₃⁻ transporters that likely play an important role in epithelial Cl⁻ absorption and HCO₃⁻ secretion. However, the mechanism of most transporters is not well understood. ***SLC26A7*** is a member of the SLC26 transporter family reported to be expressed in the basolateral membrane of the cortical collecting duct and parietal cells and functions as a coupled Cl⁻/HCO₃⁻ exchanger. In the present work we examined the transport properties of ***SLC26A7*** to determine its transport characteristics and electrogenicity. We found that when expressed in *Xenopus* oocytes or HEK293 cells ***SLC26A7*** functions as a pH_i-regulated Cl⁻ channel with minimal OH⁻/HCO₃⁻ permeability. Expression of ***SLC26A7*** in oocytes or HEK293 cells generated a Cl⁻ current with linear I/V and an instantaneous current that was voltage- and time-independent. Based on measurement of reversal potential the selectivity of ***SLC26A7*** is NO₃⁻ >> Cl⁻ = Br⁻ = I⁻ > SO₄²⁻ = Glu⁻, although I⁻ partially inhibited the current. Incubating the cells with HCO₃⁻ or butyrate acidified the cytosol and increased the selectivity of ***SLC26A7*** for Cl⁻. Measurement of membrane potential and pH_i showed minimal OH⁻ and HCO₃⁻ transport by ***SLC26A7*** when the cells were incubated in Cl⁻-containing or Cl⁻-free media. The activity of ***SLC26A7*** was inhibited by all inhibitors of anion transporters tested, 4,4'-diisothiocyanostilbene-2,2'-disulfonic acid, diphenylamine-2-carboxylic acid, and glybenclamide. These findings reveal that ***SLC26A7*** functions as a unique Cl⁻ channel that is regulated by intracellular H⁺.

ACCESSION NUMBER: 2005:204460 BIOSIS <<LOGINID::20080219>>

DOCUMENT NUMBER: PREV200500205578

TITLE: ***SLC26A7*** is a Cl⁻ channel regulated by intracellular pH.

AUTHOR(S): Kim, Kil Hwan; Shcheynikov, Nikolay; Wang, Youxue; Muallem,

Shmuel [Reprint Author]

CORPORATE SOURCE: SW Med CtrDept Physiol, Univ Texas, 5323 Harry Hines Blvd,

Dallas, TX, 75390, USA

shmuel.muallem@utsouthwestern.edu

SOURCE: Journal of Biological Chemistry, (February 25 2005)
Vol.

280, No. 8, pp. 6463-6470. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Jun 2005
Last Updated on STN: 1 Jun 2005

L1 ANSWER 39 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
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AB ***SLC26A7*** is a recently identified Cl-/HCO3- exchanger that
co-localizes with AE1 on the basolateral membrane of A intercalated
cells

(A-IC) in outer medullary collecting duct (OMCD). The purpose of
these studies was to determine whether AE1 and ***SLC26A7*** are
differentially regulated in OMCD in pathophysiologic states.

Toward this

end, the expression and regulation of AE1 and ***SLC26A7*** was
examined in water deprivation, a condition known to increase the
osmolality of the medulla. Rats were subjected to 3 d of water
deprivation while having free access to food. Northern

hybridizations

demonstrated that in the outer medulla, the mRNA expression of
SLC26A7 increased by appr300% (P < 0.01 versus control;

n = 3),

whereas the expression of AE1 decreased by appr50% (P < 0.05

versus

control, n = 3) in water-deprived rats. Immunoblot analysis

studies

demonstrated that in the outer medulla, ***SLC26A7*** abundance
increased by appr3.5-fold (P < 0.02 versus control; n = 3),

whereas the

AE1 abundance decreased by appr55% (P < 0.05 versus control) in

water

deprivation. The expression of ***SLC26A7*** remained

unchanged in

the kidney cortex and stomach in water deprivation, indicating the
specificity of ***SLC26A7*** upregulation in outer medulla. In

situ

hybridization indicated the exclusive expression of ***SLC26A7***

in

the outer medulla and double immunofluorescence labeling confirmed

the

co-localization of AE1 and ***SLC26A7*** on the basolateral

membrane

of A-IC cells in OMCD. It is concluded that AE1 and ***SLC26A7

*** are

differentially regulated in OMCD in water deprivation. On the

basis of

these results and previous functional studies indicating the

activation of

SLC26A7 activity by high osmolality, it is proposed that

SLC26A7 may play an important role in bicarbonate

reabsorption and

or cell volume regulation in OMCD (specifically under hypertonic
conditions).

ACCESSION NUMBER: 2004:397167 BIOSIS <<LOGINID::20080219>>

DOCUMENT NUMBER: PREV200400395456

TITLE: Differential regulation of basolateral Cl-/HCO3-
exchangers

SLC26A7 and AE1 in kidney outer medullary
 collecting duct.
 AUTHOR(S): Barone, Sharon; Amlal, Hassane; Xu, Jie; Kujala,
 Minna;
 Kere, Juha; Petrovic, Snezana; Soleimani, Manoocher
 [Reprint Author]
 CORPORATE SOURCE: Dept MedDiv Nephrol and Hypertens, Univ Cincinnati,
 231
 Albert Sabin Way, MSB G259, Cincinnati, OH, 45267,
 USA
 Manoocher.Soleimani@uc.edu
 SOURCE: Journal of the American Society of Nephrology,
 (August
 2004) Vol. 15, No. 8, pp. 2002-2011. print.
 CODEN: JASNEU. ISSN: 1046-6673.
 DOCUMENT TYPE: Article
 LANGUAGE: English
 ENTRY DATE: Entered STN: 13 Oct 2004
 Last Updated on STN: 13 Oct 2004

L1 ANSWER 40 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
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STN
 AB Bicarbonate transport in the collecting duct is increased post-
 natal in
 mammalian kidneys. However, little is known about the
 developmental
 regulation of the correlate molecules responsible for these
 adaptive
 changes. The purpose of these studies was to examine the
 developmental
 regulation of basolateral Cl-/HCO3- exchangers ***SLC26A7***
 and AE1,
 and the apical Cl-/HCO3-exchanger pendrin (SLC26A4) in rat kidney
 collecting duct. Accordingly, rats were studied at 3, 17 days and
 51 days
 of age. Immunofluorescence staining demonstrated that the
 SLC26A7
 labeling was barely detectable at 3 days, appeared in the inner
 medulla at
 17 days and achieved adult expression levels in collecting duct at
 51
 days. The expression of pendrin in β-intercalated cells of
 CCD was
 very low at 3 days, increased at 17 days and achieved adult
 expression
 levels at 51 days of age. AE1 labeling was detected at 3 days and
 increased progressively at 17 and 51 days of age. In conclusion,
 our
 results demonstrate a maturation dependent enhancement of
 expression of
 basolateral (***SLC26A7*** and AE1) and apical (pendrin)
 Cl-/HCO3-
 exchangers in the collecting duct. These results are consistent
 with
 functional studies indicating age dependent upregulation of
 bicarbonate
 transport in the collecting duct. This study was supported
 through the
 Veterans Affairs Merit Award to MS and the award from Kidney
 Foundation of

Greater Cincinnati to SP.

ACCESSION NUMBER: 2004:289133 BIOSIS <<LOGINID::20080219>>
DOCUMENT NUMBER: PREV200400287890
TITLE: Developmental regulation of Cl-/HCO3- exchangers in the kidney collecting duct.

AUTHOR(S): Petrovic, Snezana [Reprint Author]; Ma, Liyun; Barone, Sharon; Kujala, Minna; Kere, Juha; Soleimani, Manoocher

CORPORATE SOURCE: Internal Medicine, University of Cincinnati, 231 Albert Sabin Way, Cincinnati, OH, 45267-0585, USA
snezana.petrovic@med.va.gov

SOURCE: FASEB Journal, (2004) Vol. 18, No. 4-5, pp. Abst. 215.6.
<http://www.fasebj.org/>. e-file.
Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. Washington, District of Columbia, USA. April 17-21, 2004. FASEB. ISSN: 0892-6638 (ISSN print).

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 16 Jun 2004
Last Updated on STN: 16 Jun 2004

L1 ANSWER 41 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AB The outer medullary collecting duct (OMCD) plays an important role in bicarbonate reabsorption and acid-base regulation. An apical V-type H+-ATPase and a basolateral Cl-/HCO3- exchanger, located in intercalated cells of OMCD, mediate the bicarbonate reabsorption. Here we report the identification of a new basolateral Cl-/HCO3- exchanger in OMCD intercalated cells in rat kidney. Northern hybridizations demonstrated the predominant expression of this transporter, also known as ***SLC26A7***, in the outer medulla, with lower expression levels in the inner medulla. ***SLC26A7*** was recognized as a approx90-kDa band in the outer medulla by immunoblot analysis and was localized on the basolateral membrane of a subset of OMCD cells by immunocytochemical staining. No labeling was detected in the cortex. Double-immunofluorescence labeling with the aquaporin-2 and ***SLC26A7*** antibodies or anion exchanger-1 and ***SLC26A7*** antibodies identified the ***SLC26A7***-expressing cells as alpha-intercalated cells. Functional studies in oocytes demonstrated that increasing the osmolality of the media (to simulate the physiological milieu in the

medulla) increased the Cl-/HCO3- exchanger activity mediated via
 SLC26A7 by about threefold (P<0.02 vs. normal condition).
 We
 propose that ***SLC26A7*** is a basolateral Cl-/HCO3-exchanger
 in
 intercalated cells of the OMCD and may play an important role in
 bicarbonate reabsorption in medullary collecting duct.
 ACCESSION NUMBER: 2004:172857 BIOSIS <<LOGINID::20080219>>
 DOCUMENT NUMBER: PREV200400174436
 TITLE: ***SLC26A7*** : A basolateral Cl-/HCO3- exchanger
 specific to intercalated cells of the outer
 medullary
 collecting duct.
 AUTHOR(S): Petrovic, Snezana; Barone, Sharon; Xu, Jie;
 Conforti,
 Laura; Ma, Liyun; Kujala, Minna; Kere, Juha;
 Soleimani,
 Manoocher [Reprint Author]
 CORPORATE SOURCE: Division of Nephrology and Hypertension, Dept. of
 Medicine,
 Univ. of Cincinnati, 231 Albert Sabin Way, MSB G259,
 Cincinnati, OH, 45267-0585, USA
 Manoocher.Soleimani@uc.edu
 SOURCE: American Journal of Physiology, (January 2004) Vol.
 286,
 No. 1 Part 2, pp. F161-F169. print.
 ISSN: 0002-9513 (ISSN print).
 DOCUMENT TYPE: Article
 LANGUAGE: English
 ENTRY DATE: Entered STN: 31 Mar 2004
 Last Updated on STN: 31 Mar 2004

L1 ANSWER 42 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
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ACCESSION NUMBER: 2004:109854 BIOSIS <<LOGINID::20080219>>
 DOCUMENT NUMBER: PREV200400106688
 TITLE: Anion transporters SLC26A6 and ***SLC26A7*** are
 coexpressed in collecting ducts of human kidney.
 AUTHOR(S): Kujala, Minna [Reprint Author]; Tienari, Jukka;
 Lohi,
 Hannes [Reprint Author]; Lehtonen, Eero; Kere, Juha
 [Reprint Author]
 CORPORATE SOURCE: Department of Medical Genetics, Biomedicum,
 University of
 Helsinki, Helsinki, Finland
 SOURCE: Journal of the American Society of Nephrology,
 (November
 2003) Vol. 14, No. Abstracts Issue, pp. 775A. print.
 Meeting Info.: Meeting of the American Society of
 Nephrology Renal Week. San Diego, CA, USA. November
 12-17,
 2003. American Society of Nephrology.
 CODEN: JASNEU. ISSN: 1046-6673.
 DOCUMENT TYPE: Conference; (Meeting)
 Conference; (Meeting Poster)
 Conference; Abstract; (Meeting Abstract)
 LANGUAGE: English
 ENTRY DATE: Entered STN: 25 Feb 2004
 Last Updated on STN: 25 Feb 2004

L1 ANSWER 43 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:109651 BIOSIS <<LOGINID::20080219>>
DOCUMENT NUMBER: PREV200400106580
TITLE: Differential regulation of basolateral Cl-/HCO3-exchangers
AE1 and ***SLC26A7*** in outer medullary collecting duct.

AUTHOR(S): Barone, Sharon [Reprint Author]; Amlal, Hassane [Reprint Author]; Xu, Jie [Reprint Author]; Soleimani, Manoocher [Reprint Author]

CORPORATE SOURCE: Department of Medicine, University of Cincinnati, Cincinnati, OH, USA
SOURCE: Journal of the American Society of Nephrology, (November 2003) Vol. 14, No. Abstracts Issue, pp. 541A. print. Meeting Info.: Meeting of the American Society of Nephrology Renal Week. San Diego, CA, USA. November 12-17, 2003. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)

LANGUAGE: English
ENTRY DATE: Entered STN: 25 Feb 2004
Last Updated on STN: 25 Feb 2004

L1 ANSWER 44 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:92438 BIOSIS <<LOGINID::20080219>>
DOCUMENT NUMBER: PREV200400085630
TITLE: Identification of a basolateral Cl-/HCO3- exchanger specific to - intercalated cells in outer medullary collecting duct: Co-localization with AE1.

AUTHOR(S): Petrovic, Snezana [Reprint Author]; Barone, Sharon [Reprint Author]; Xu, Jie [Reprint Author]; Ma, Liyun [Reprint Author]; Conforti, Laura [Reprint Author]; Kujala, Mina; Kere, Juha; Soleimani, Manoocher [Reprint Author]

CORPORATE SOURCE: Medicine, University of Cincinnati, Cincinnati, OH, USA
SOURCE: Journal of the American Society of Nephrology, (November 2003) Vol. 14, No. Abstracts Issue, pp. 68A. print. Meeting Info.: Meeting of the American Society of Nephrology Renal Week. San Diego, CA, USA. November 12-17, 2003. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English
ENTRY DATE: Entered STN: 11 Feb 2004

Last Updated on STN: 11 Feb 2004

L1 ANSWER 45 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
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STN

ACCESSION NUMBER: 2004:92224 BIOSIS <<LOGINID::20080219>>
DOCUMENT NUMBER: PREV200400085417
TITLE: Immunolocalization of anion exchanger ***SLC26A7
*** in
mouse kidney.
AUTHOR(S): Dudas, Paul L. [Reprint Author]; Greineder, Colin F.
[Reprint Author]; Mentone, SueAnn [Reprint Author];
Aronson, Peter S. [Reprint Author]
CORPORATE SOURCE: Depts. of Medicine and Physiology, Yale School of
Medicine,
New Haven, CT, USA
SOURCE: Journal of the American Society of Nephrology,
(November
2003) Vol. 14, No. Abstracts Issue, pp. 313A. print.
Meeting Info.: Meeting of the American Society of
Nephrology Renal Week. San Diego, CA, USA. November
12-17,
2003. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Feb 2004
Last Updated on STN: 11 Feb 2004

L1 ANSWER 46 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
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STN

AB The basolateral Cl-/HCO3- exchanger in parietal cells plays an
essential
role in gastric acid secretion that is mediated via the apical
gastric
H-K-ATPase. Here we report the identification of a new Cl-/HCO3-
exchanger, which shows exclusive expression in mouse stomach and
kidney,
with expression in the stomach limited to the basolateral membrane
of
gastric parietal cells. Tissue distribution studies by RT-PCR and
Northern hybridizations demonstrated the exclusive expression of
this
transporter, also known as ***SLC26A7***, to stomach and
kidney, with
the stomach expression significantly more abundant. No expression
was
detected in the intestine. Cellular distribution studies by RT-PCR
and
Northern hybridizations demonstrated predominant localization of
SLC26A7 in gastric parietal cells. Immunofluorescence
labeling
localized this exchanger exclusively to the basolateral membrane of
gastric parietal cells, and functional studies in oocytes indicated
that
SLC26A7 is a DIDS-sensitive, Cl-/HCO3- exchanger.
SLC26A7
is active at both acidic and alkaline pH, a property distinct from

AE2
which is inactive at acidic pH. With respect to the activation at
the
acidic pH, ***SLC26A7*** exhibits a pH profile similar to the
basolateral Cl-/HCO3- exchanger in native parietal cells. Based on
its
unique expression pattern and function, we propose that
SLC26A7
is a basolateral Cl-/HCO3- exchanger in gastric parietal cells and
plays a
major role in gastric acid secretion..

ACCESSION NUMBER: 2004:34249 BIOSIS <<LOGINID::20080219>>
DOCUMENT NUMBER: PREV200400032337
TITLE: IDENTIFICATION OF A BASOLATERAL CL-/HCO3- EXCHANGER
SPECIFIC TO GASTRIC PARIETAL CELLS.
AUTHOR(S): Petrovic, Snezana [Reprint Author]; Ju, Xie; Barone,
Sharon; Seidler, Ursula; Kere, Juha; Soleimani,
Manoocher
CORPORATE SOURCE: Cincinnati, OH, USA
SOURCE: Digestive Disease Week Abstracts and Itinerary
Planner,
(2003) Vol. 2003, pp. Abstract No. 32. e-file.
Meeting Info.: Digestive Disease 2003. FL, Orlando,
USA.
May 17-22, 2003. American Association for the Study
of
Liver Diseases; American Gastroenterological
Association;
American Society for Gastrointestinal Endoscopy;
Society
for Surgery of the Alimentary Tract.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Jan 2004
Last Updated on STN: 7 Jan 2004

L1 ANSWER 47 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
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STN

AB The basolateral Cl-/HCO3- exchanger in parietal cells plays an
essential
role in gastric acid secretion mediated via the apical gastric
H+-K+-ATPase. Here, we report the identification of a new
Cl-/HCO3-
exchanger, which shows exclusive expression in mouse stomach and
kidney,
with expression in the stomach limited to the basolateral membrane
of
gastric parietal cells. Tissue distribution studies by RT-PCR and
Northern hybridizations demonstrated the exclusive expression of
this
transporter, also known as ***SLC26A7***, to stomach and
kidney, with
the stomach expression significantly more abundant. No expression
was
detected in the intestine. Cellular distribution studies by RT-PCR
and
Northern hybridizations demonstrated predominant localization of
SLC26A7 in gastric parietal cells. Immunofluorescence
labeling

localized this exchanger exclusively to the basolateral membrane of gastric parietal cells, and functional studies in oocytes indicated that

SLC26A7 is a DIDS-sensitive Cl-/HCO3- exchanger that is active in

both acidic and alkaline pH. On the basis of its unique expression

pattern and function, we propose that ***SLC26A7*** is a basolateral

Cl-/HCO3- exchanger in gastric parietal cells and plays a major role in

gastric acid secretion.

ACCESSION NUMBER: 2003:404271 BIOSIS <<LOGINID::20080219>>

DOCUMENT NUMBER: PREV200300404271

TITLE: Identification of a basolateral Cl-/HCO3- exchanger specific to gastric parietal cells.

AUTHOR(S): Petrovic, Snezana; Ju, Xie; Barone, Sharon; Seidler, Ursula; Alper, Seth L.; Lohi, Hannes; Kere, Juha; Soleimani, Manoocher [Reprint Author]

CORPORATE SOURCE: Div. of Nephrology and Hypertension, Dept. of Medicine,

Univ. of Cincinnati, 231 Albert Sabin Way, MSB G259, Cincinnati, OH, 45267-0585, USA
Manoocher.Soleimani@uc.edu

SOURCE: American Journal of Physiology, (June 2003) Vol. 284, No. 6

Part 1, pp. G1093-G1103. print.
ISSN: 0002-9513 (ISSN print).

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 3 Sep 2003

Last Updated on STN: 3 Sep 2003

L1 ANSWER 48 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:166591 BIOSIS <<LOGINID::20080219>>

DOCUMENT NUMBER: PREV200300166591

TITLE: Anion exchangers SLC26A6 and ***SLC26A7*** are expressed in collecting ducts of human kidney.

AUTHOR(S): Kujala, M. M. [Reprint Author]; Tienari, J.; Lohi, H.

[Reprint Author]; Lehtonen, E.; Kere, J. [Reprint

Author]

CORPORATE SOURCE: Department of Medical Genetics, Biomedicum, University of

Helsinki, Helsinki, Finland

SOURCE: Molecular Biology of the Cell, (Nov 2002) Vol. 13, No.

Supplement, pp. 269a. print.

Society

Meeting Info.: 42nd Annual Meeting of the American

for Cell Biology. San Francisco, CA, USA. December

14-18,

2002. American Society for Cell Biology.

ISSN: 1059-1524 (ISSN print).

DOCUMENT TYPE: Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 2 Apr 2003

Last Updated on STN: 2 Apr 2003

L1 ANSWER 49 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
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STN

AB Congenital chloride diarrhea (CLD) is an autosomal recessive disorder of intestinal electrolyte absorption. It is characterized by persistent secretory diarrhea resulting in polyhydramnios and prematurity prenatally, and dehydration, hyponatremia, hyperbilirubinemia, abdominal distention, and failure to thrive immediately after birth. CLD is caused by mutations in the solute carrier family 26, member 3 gene (SLC26A3, alias CLD or DRA), which encodes a Na⁺ independent Cl⁻/HCO₃⁻ (or OH⁻) exchanger. SLC26A3 is a member of the SLC26 sulfate permease/anion transporter family and it is expressed mainly in the apical brush border of intestinal epithelium. The only extraintestinal tissues showing SLC26A3 expression are eccrine sweat glands and seminal vesicles. A wide variety of different mutations in the SLC26A3 gene have been associated with CLD with no apparent evidence of phenotype-genotype correlation. The clinical course of CLD, however, is variable and may rather depend on environmental factors and compensatory mechanisms than mutations. In this report, we present a summary of all published and two novel SLC26A3 mutations and polymorphisms, and review them in the context of their functional consequences and clinical implications.

ACCESSION NUMBER: 2003:47093 BIOSIS <<LOGINID::20080219>>
DOCUMENT NUMBER: PREV200300047093
TITLE: SLC26A3 mutations in congenital chloride diarrhea.
AUTHOR(S): Makela, Siru; Kere, Juha; Holmberg, Christer; Hoglund, Pia
[Reprint Author]
CORPORATE SOURCE: Hospital for Children and Adolescents, Department of Pediatrics, University of Helsinki, Stenbackinkatu11, P.O. Box 281, Helsinki, 00029 HUS, Finland
PiaHoglund@Helsinki.Fi
SOURCE: Human Mutation, (December 2002) Vol. 20, No. 6, pp. 425-438. print.
ISSN: 1059-7794.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
OTHER SOURCE: DDBJ-AC002467; EMBL-AC002467; GenBank-AC002467; GenBank-AC005064; GenBank-AF030880; GenBank-AF279265; GenBank-AF297659; GenBank-AF331521; GenBank-AF331522; GenBank-AF331523; GenBank-AF331524; GenBank-AF331525; GenBank-L02785; GenBank-U14528
ENTRY DATE: Entered STN: 15 Jan 2003

Last Updated on STN: 4 Mar 2003

L1 ANSWER 50 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
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ACCESSION NUMBER: 2002:567169 BIOSIS <<LOGINID::20080219>>
DOCUMENT NUMBER: PREV200200567169
TITLE: ***SLC26A7*** is an apical Cl⁻/HCO₃⁻ exchanger
in
stomach mucous cells and is regulated by gastric
acid.
AUTHOR(S): Barone, Sharone [Reprint author]; Ju, Xie [Reprint
author];
Petrovic, Snezana [Reprint author]; Soleimani,
Manoocher
[Reprint author]
CORPORATE SOURCE: Internal Medicine, University of Cincinnati,
Cincinnati,
OH, USA
SOURCE: Journal of the American Society of Nephrology,
(September,
2002) Vol. 13, No. Program and Abstracts Issue, pp.
63A.
print.
Meeting Info.: Meeting of the American Society of
Nephrology. Philadelphia, PA, USA. October 30-
November 04,
2002. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Nov 2002
Last Updated on STN: 7 Nov 2002

L1 ANSWER 51 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
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STN

AB A second distinct family of anion exchangers, SLC26, in addition to
the
classical SLC4 (or anion exchanger) family, has recently been
delineated.
Particular interest in this gene family is stimulated by the fact
that the
SLC26A2, SLC26A3, and SLC26A4 genes have been recognized as the
disease
genes mutated in diastrophic dysplasia, congenital chloride
diarrhea, and
Pendred syndrome, respectively. We report the expansion of the
SLC26 gene
family by characterizing three novel tissue-specific members, named
SLC26A7, SLC26A8, and SLC26A9, on chromosomes 8, 6, and
1,
respectively. The ***SLC26A7*** -A9 proteins are structurally
very
similar at the amino acid level to the previous family members and
show
tissue-specific expression in kidney, testis, and lung,
respectively.
More detailed characterization by immunohistochemistry and/or in
situ

hybridization localized ***SLC26A7*** to distal segments of nephrons, SLC26A8 to developing spermatocytes, and SLC26A9 to the luminal side of the bronchiolar and alveolar epithelium of lung. Expression of ***SLC26A7*** -A9 proteins in Xenopus oocytes demonstrated chloride, sulfate, and oxalate transport activity, suggesting that they encode functional anion exchangers. The functional characterization of the novel tissue-specific members may provide new insights to anion transport physiology in different parts of body.

ACCESSION NUMBER: 2002:420206 BIOSIS <<LOGINID::20080219>>

DOCUMENT NUMBER: PREV200200420206

TITLE: Functional characterization of three novel tissue-specific

anion exchangers ***SLC26A7*** , -A8, and -A9.
AUTHOR(S): Lohi, Hannes; Kujala, Minna; Makela, Siru; Lehtonen, Eero;

Kestila, Marjo; Saarialho-Kere, Ulpu; Markovich, Daniel;

Kere, Juha [Reprint author]
CORPORATE SOURCE: Dept. of Biosciences at Novum, Karolinska Institute, 14157,

Huddinge, Sweden
juha.kere@biosci.ki.se
SOURCE: Journal of Biological Chemistry, (April 19, 2002)
Vol. 277,

No. 16, pp. 14246-14254. print.
CODEN: JBCHA3. ISSN: 0021-9258.

DOCUMENT TYPE: Article

LANGUAGE: English

OTHER SOURCE: Genbank-AC005064; Genbank-AC017061; Genbank-AF030880;

Genbank-AF230376; Genbank-AF279265; Genbank-AF297659;

Genbank-AF331521; Genbank-AF331522; Genbank-AF331523;

Genbank-AF331524; Genbank-AF331525; Genbank-AL133507;

Genbank-AL360009; Genbank-L02785; Genbank-U14528
ENTRY DATE: Entered STN: 7 Aug 2002

Last Updated on STN: 23 Sep 2002

L1 ANSWER 52 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on

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AB The SLC26 gene family encodes anion exchangers involved in the transport of halides, bicarbonate, SO42-, formate, and oxalate. We report the cloning and characterization of 4 new family members, in both mouse and human. The human ***SLC26A7*** , A8, A9, and A11 genes are found on chromosomes 8q24, 6p21, 1p31, and 17q24, respectively. A11 is ubiquitous, the others are more restricted in expression; A7 in renal papilla, A8 in brain and testis, and A9 in lung. The predicted proteins vary in

length
from 656 (A7) to 970 (A8) amino acids, each with a central
hydrophobic
"sulfate transporter" domain and a C-terminal STAS domain. The
comparison
of A1 (Sat-1), A2 (DTDST), A6, A7, and A9 in *Xenopus* oocytes
reveals
marked functional heterogeneity. SLC26A9 is thus highly selective,
transporting Cl⁻ but not formate, oxalate, or SO₄²⁻; measurement of
intracellular pH in the presence of HCO₃⁻ indicates that it
functions as a
Cl⁻-base exchanger. The effect of acid-outside pH gradient on Cl⁻-
Cl⁻
exchange is heterogeneous; A2 is inhibited, A6 is unaffected, and
A9 is
activated. The cloning of new SLC26 genes promises to yield new
insights
in the molecular physiology of anion transport.

ACCESSION NUMBER: 2002:369431 BIOSIS <<LOGINID::20080219>>

DOCUMENT NUMBER: PREV200200369431

TITLE: Cloning of multiple novel members of the SLC26 gene
family

of anion exchangers.

AUTHOR(S): Xie, Qizhi [Reprint author]; Welch, Rick [Reprint
author];

Song, Luyan [Reprint author]; Romero, Michael F.;

Mount,

David B.

CORPORATE SOURCE: Division of Nephrology, Vanderbilt University
Medical

Center, S-3223 MCN, VUMC, Nashville, TN, 37232, USA
FASEB Journal, (March 22, 2002) Vol. 16, No. 5, pp.

SOURCE:
A807.

print.

Meeting Info.: Annual Meeting of Professional

Research

Scientists on Experimental Biology. New Orleans,

Louisiana,

USA. April 20-24, 2002.

CODEN: FAJOEC. ISSN: 0892-6638.

DOCUMENT TYPE:

Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE:

English

ENTRY DATE:

Entered STN: 3 Jul 2002

Last Updated on STN: 3 Jul 2002

L1 ANSWER 53 OF 134 BIOSIS COPYRIGHT (c) 2008 The Thomson
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STN

AB A unique characteristic of endothelial cells from high endothelial
venules

(HEVEC) in lymphoid organs and chronically inflamed tissues is
their

capacity to incorporate large amounts of sulfate into sialomucin-
type

counter-receptors for the lymphocyte homing receptor L-selectin.
We have

previously shown that HEVEC express two functional classes of
sulfate

transporters: sodium/sulfate cotransporters and sulfate/anion
exchangers.

Here, we report the molecular cloning from human HEVEC of a 2.9-kb cDNA

encoding ***SLC26A7***, a novel member of the SLC26 (solute carrier 26) sulfate/anion exchanger family. ***SLC26A7*** exhibits 30% identity with three known sulfate transporters from the SLC26 family:

SLC26A2 (also known as DTDST), SLC26A1 (also known as SAT1), and SLC26A3

(also known as DRA). Northern blot analysis revealed specific expression

of ***SLC26A7*** mRNA in kidney. Alternative splicing and polyadenylation of ***SLC26A7*** pre-mRNA in kidney suggest the existence of two protein isoforms, ***SLC26A7***.1 and ***SLC26A7***

.2, differing in their carboxy termini.

ACCESSION NUMBER: 2002:182547 BIOSIS <<LOGINID::20080219>>

DOCUMENT NUMBER: PREV200200182547

TITLE: Molecular cloning of ***SLC26A7***, a novel member of

the SLC26 sulfate/anion transporter family, from high

endothelial venules and kidney.

AUTHOR(S): Vincourt, Jean-Baptiste; Jullien, Denis; Kossida, Sophia;

Amalric, Francois; Girard, Jean-Philippe [Reprint author]

CORPORATE SOURCE: Laboratoire de Biologie Vasculaire, Institut de Pharmacologie et de Biologie Structurale du CNRS, 205 Route

de Narbonne, 31077, Toulouse, France
girard@ipbs.fr

SOURCE: Genomics, (February, 2002) Vol. 79, No. 2, pp. 249-256.

print.

CODEN: GNMCEP. ISSN: 0888-7543.

DOCUMENT TYPE: Article

LANGUAGE: English

OTHER SOURCE: Genbank-AAK19153; Genbank-AAK95666; Genbank-NP000432;

Genbank-NP11467; Genbank-P50443; Genbank-XP004952; Genbank-XP011158

ENTRY DATE: Entered STN: 6 Mar 2002

Last Updated on STN: 6 Mar 2002

L1 ANSWER 54 OF 134 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation
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AB In the present study, the effect of potassium depletion on the expression of acid-base transporters in the collecting duct was examined.

Toward this end rats were fed a potassium-free diet for 3 weeks. Thereafter, the expression of the basolateral chloride/bicarbonate exchangers AE1 and ***SLC26A7*** and the apical H⁺-ATPase was examined

by northern hybridization, immunoblot analysis and immunofluorescence

labelling. The mRNA expression of AE1 increased by a robust similar to

500% in the cortex and similar to 70% in the outer medulla, which translated into a huge increase in AE1 protein abundance in the

cortex and
a moderate increase in the outer medulla in K-depletion. The mRNA
expression of ***SLC26A7*** did not change significantly but
its
protein abundance showed a robust increase in the outer medulla.
The
expression of ***SLC26A7*** remained undetected in the cortex
in
K-depleted rats. The post translational increase in ***SLC26A7

membrane abundance in potassium depletion was recapitulated in
vitro using
epitope-tagged ***SLC26A7***. H⁺-ATPase displayed enhanced
apical
plasma membrane immunoreactivity in the OMCD in K-depletion. We
suggest
that the up-regulation of ***SLC26A7*** and AE1 on the
basolateral
membrane of A-intercalated cells in the OMCD and CCD, respectively,
along
with H⁺-ATPase on the apical membrane, contributes to enhanced
bicarbonate
absorption in the collecting duct in K-depletion.

ACCESSION NUMBER: 2008:11966 SCISEARCH <<LOGINID::20080219>>
THE GENUINE ARTICLE: 236XJ
TITLE: Regulation of the basolateral chloride/base
exchangers AE1
and ***SLC26A7*** in the kidney collecting duct
in
potassium depletion

AUTHOR: Barone, Sharon; Amlal, Hassané; Kujala, Minna; Xu,
Jie;
Karet, Fiona; Blanchard, Ann; Kere, Juha;
Soleimani,
Manoocher (Reprint)

CORPORATE SOURCE: Univ Cincinnati, Dept Med, 231 Albert Sabin Way,
MSB G259,
Cincinnati, OH 45267 USA (Reprint); Univ
Cincinnati, Dept
Med, Cincinnati, OH 45267 USA; Univ Helsinki, FIN-
00014
Helsinki, Finland; Univ Cambridge, Dept Med Genet,
Div
Renal Med, Cambridge, England; Univ Paris 05, Fac
Med,
Paris, France; Karolinska Inst, Huddinge, Sweden;
Vet
Affairs Med Ctr, Res Serv, Cincinnati, OH USA
manoocher.soleimani@uc.edu

COUNTRY OF AUTHOR: USA; Finland; England; France; Sweden
SOURCE: NEPHROLOGY DIALYSIS TRANSPLANTATION, (DEC 2007)
Vol. 22,
No. 12, pp. 3462-3470.
ISSN: 0931-0509.

PUBLISHER: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD OX2
6DP,
ENGLAND.

DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 40
ENTRY DATE: Entered STN: 3 Jan 2008

L1 ANSWER 55 OF 134 SCISEARCH COPYRIGHT (c) 2008 The Thomson
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on STN

AB To date three potential candidates for parietal cell
basolateral Cl-
entry have been described: the highly 4,4'-
diisothiocyanatostilbene-2,2'-
disulfonic acid (DIDS)-sensitive Cl-/HCO₃ exchanger AE2, the HCO₃-
and
lowly DIDS-sensitive ***SLC26A7*** protein, and the Na⁺-2Cl⁻(-)K
(+) cotransporter (NKCC1). In this study we investigate the
contribution of
these pathways to secretagogue stimulated acid secretion.
Individually
hand-dissected rat gastric glands were microfluorimetrically
monitored for
Cl- influx and pH(i) changes. Transporter activity was determined
by
varying ion content and through the use of pharmacological
inhibitors.
Expression of ***SLC26A7*** in rat parietal cells was shown by
immunohistochemistry and Western blot. ***SLC26A7*** was
inhibited by
5-Nitro-2-(3-phenylpropyl-amino)benzoic acid (NPPB) (100 mu M) in
the
Xenopus laevis oocyte expression system. Cl- influx in parietal
cells was
enhanced by histamine, depended partially on endogenous HCO₃-
synthesis
and completely on extracellular Na⁺. Removal and subsequent
readdition of
Cl- revealed a low and a high DIDS-sensitive HCO₃- extrusion system
contributing to Cl- uptake. At acidic pH(i), however, H⁺ extrusion
via
the H⁺,K⁺-ATPase depending on Cl- uptake was abolished only in the
presence of 100 mu M (NPPB) and at high (250 mu M) DIDS
concentration.
There was no effect of the NKCC inhibitor bumetanide on stimulated
H⁺
extrusion. These results would be compatible with ***SLC26A7***
as a

Cl- uptake system under histamine stimulation.

ACCESSION NUMBER: 2007:985171 SCISEARCH <<LOGINID::20080219>>

THE GENUINE ARTICLE: 205NX

TITLE: ***SLC26A7*** Can function as a chloride-
loading

mechanism in parietal cells

AUTHOR: Kosiek, Ortrud; Busque, Stephanie M.; Foeller,
Michael;

Shcheynikov, Nikolay; Kirchhoff, Philipp; Bleich,
Markus;

Muallem, Shmuel; Geibel, John P. (Reprint)
CORPORATE SOURCE: Yale Univ, Sch Med, Dept Surg, BML 265, 310 Cedar
St, New

Haven, CT 06520 USA (Reprint); Yale Univ, Sch Med,
Dept
Surg, New Haven, CT 06520 USA; Univ Kiel, Inst

Physiol,
 Transplantat
 Dept
 Dept
 Kiel, Germany; Univ Zurich, Dept Visceral &
 Surg, Zurich, Switzerland; Univ Texas, SW Med Ctr,
 Physiol, Dallas, TX 75235 USA; Yale Univ, Sch Med,
 Cellular & Mol Physiol, New Haven, CT 06510 USA
 john.geibel@yale.edu
 COUNTRY OF AUTHOR: USA; Germany; Switzerland
 SOURCE: PFLUGERS ARCHIV-EUROPEAN JOURNAL OF PHYSIOLOGY,
 (SEP 2007) Vol. 454, No. 6, pp. 989-998.
 ISSN: 0031-6768.
 PUBLISHER: SPRINGER, 233 SPRING STREET, NEW YORK, NY 10013
 USA.
 DOCUMENT TYPE: Article; Journal
 LANGUAGE: English
 REFERENCE COUNT: 35
 ENTRY DATE: Entered STN: 4 Oct 2007
 Last Updated on STN: 4 Oct 2007
 ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L1 ANSWER 56 OF 134 SCISEARCH COPYRIGHT (c) 2008 The Thomson
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AB Aims: Anions have an important role in the regulation of
 airway
 surface liquid (ASL) volume, viscosity and pH. However, functional
 localization and regulation of anion exchangers (AEs) have not been
 clearly described. The aim of this study was to investigate the
 regulation of AE mRNA expression level in accordance with
 mucociliary
 differentiation and the functional expression of AEs cultured
 normal human
 nasal epithelial (NHNE) cells.
 Methods: Nasal mucosal specimens from three patients are
 obtained and
 serially cultured cells are subjected to morphological
 examinations,
 RT-PCR, Western blot analysis and immunocytochemistry. AE activity
 is
 assessed by pH_i measurements.
 Results: Expression of ciliated cells on the apical membrane
 and
 expression of MUC5AC, a marker of mucous differentiation, increased
 with
 time. AE2 and SLC26A4 mRNA expression decreased as mucociliary
 differentiation progressed, and AE4, ***SLC26A7*** and SLC26A8
 mRNA
 expression increased on the 14th and 28th day after confluence.
 Accordingly, AE4 protein expression also progressively increased.
 AE
 activity in 100 mM K⁺ buffer solutions was nearly twofold higher
 than that
 in 5 mM K⁺ buffer solutions. Moreover, only luminal AE activity
 increased
 about fourfold over the control in the presence of 5 μM
 forskolin. In
 the presence of 100 μM adenosine-5'-triphosphate (ATP) which
 evokes

intracellular calcium signalling through activation of purinergic receptors, only luminal AE activity was again significantly increased. On the other hand, 500 μ M 4,4'-diisothiocyanostilbene-2,2'-disulfonic acid (DIDS), an inhibitor of most SLC4 and SLC26AE isoforms, nearly abolished AE activity in both luminal and basolateral membranes. We found that AE activity was affected by intracellular cAMP and calcium signalling in the luminal membrane and was DIDS-sensitive in both membranes of cultured NHNE cells.

Conclusion: Our findings through molecular and functional studies using cultured NHNE cells suggest that AEs may have an important role in the regulation of ASL.

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